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A CONTRIBUTION TO THE STUDY OF SYRINGOMYELIA (HYDROMYELIA).

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It is well known that a dilatation of the central canal is not infrequently found in the fœtal spinal cord, especially in connection with other developmental irregularities of the cord. Its appearance in the adult is less common, and when found, it is often by accident, no symptoms having occurred during life to call attention to the cord. Not in all cases, however, in which the central canal of the adult cord has appeared enlarged, has the condition been one of simple dilatation (internal hydromyelia), as in the fœtus; most cases present, on the contrary, peculiar complications, which render it doubtful whether the cavities found represent congenital dilatation of the canal, or whether they are to be attributed to pathological changes which have occurred in later life.

The chief circumstances complicating the simple cavity-formation are a thickening of the tissues surrounding the cavity, and the appearance of new growths. There may also be grey degeneration (sclerosis) of the posterior columns, atrophy of the grey substance, hæmorrhages in or about the cavity, and sometimes the remains of acute or chronic circumscribed myelitis in the cord or medulla oblongata. Such pathological appearances can be attributed partly to

the direct mechanical pressure of the distended cavity, and partly to inflammatory changes resulting from this pressure. In order to arrive at the right interpretation of the cavities and their mode of origin, those cases only have to be considered in which the formation of the cavity is complicated with the growth of a neoplasm, or with a thickening of the tissues around the cavity.

Hallopeau [1] had already in 1870 attempted to explain the formation of cavities in certain cases, otherwise than by a congenital dilatation of the central canal. The thickening of the surrounding tissues (sclérose diffuse periépendymaire) he assumed to indicate an inflammatory process occurring in later life; and thought that the new growth resulting from the inflammation could partly thicken and partly soften. The central canal, according to him, remains in parts unaltered, in others becomes dilated and destroyed, on account of the softening of the surrounding tissue, and of the pressure of the serum poured into its lumen; whilst the newly-formed connective tissue persists and increases. The central canal might thus, he thought, become irregular, extending backwards and partly losing its epithelium; while, on the other hand, its lumen might be encroached upon by the increasing tissue of the ependyma.

The question was revived by the studies of Simon [2] (who at the same time critically reviewed the earlier literature), and by my own investigations.[3] Simon, as well as myself, was able to distinguish a normal central canal in its usual position and *in front of* the cavity, from which we drew the conclusion that the latter could not represent a dilatation of the former. We found also in each case a new growth, as well as a cavity in the spinal cord, with a probable connection between the cavity and the new growth, and we both endeavoured to show that the cavity was merely the result of a softening of the tumour beginning in its central part. This view

[1] "Contribution à l'étude de la sclérose diffuse périépendymaire;" 'Gaz. Méd.' 1870, Nos. 30, 32, 34, 35. "Etude sur les myélites chroniques diffuses;" 'Arch. génér. de Méd.' 1871, 1872.

[2] Th. Simon, "Beiträge zur Pathologie und pathol. Anatomie des Central-Nervensystems;" 'Arch. für Psych. und Nervenkrankheiten,' V. i. 1874, p. 109.

[3] Westphal, "Ueber einen Fall von Höhlen und Geschwulstbildung im Rückenmark," &c.; ibid. p. 30.

was strengthened by the fact that, in certain sections, parts of the new growth were still recognisable on the inner wall of the cavity. One can see plainly in the illustration accompanying my former article (Plate III.) that there exists a large cavity in the upper part of the cord, surrounded by a thick capsule; lower down portions of new growth appear on its inner wall; and still lower, a consistent tumour, in which only a small canal is visible. In front of the cavity and tumour appears the normal central canal in its usual position. What supposition could in this case be more probable than that the original pathological condition was a new growth, which was gradually disintegrated from above downwards, and so led to the formation of a cavity?

It is nevertheless remarkable that in most recorded cases the seat of the lesion is constant, being generally found in the *posterior columns* of the cord; further, that, apart from the central canal, cavities are found in the new growth which show the remains of a lining of *cylindrical epithelium*. Although Simon was inclined to draw the conclusion that the cavity formed by the softening and absorption of a new growth could under certain circumstances become lined with cylindrical epithelium—a view not supported by other facts—the thought suggests itself, on the other hand, that we have to do either with congenital anomalies in the spinal cord, such as a double or triple central canal, or with a constriction of the canal by the pushing in of a new growth. That a divided central canal may occur in a spinal cord presenting no further anomaly, is a fact sufficiently evident, however we may explain its existence, so that the supposition of Simon to explain the occasional lining of the cavities in cases of syringomyelia with cylindrical epithelium, is unnecessary.[1]

Finally, embryology has taught us how it is that the cavity almost unexceptionally has its seat in the posterior columns; for at a certain stage of development the central canal extends as a fissure between the posterior columns, to the periphery of the cord, and becomes enclosed at a later

[1] Compare, amongst others, Pick, "Ueber die Entstehung eines mehrfachen Central-Kanal;" 'Arch. für Psych. und Nervenkrankheiten,' VIII. 2, 1878, p. 285. Pick describes here a triple central canal. I have myself recently seen a similar case. Compare 'Charité Annalen,' 1883.

period by the transverse development of the posterior columns. If this development is interrupted and the fissure is only partly obliterated, there will remain a cavity or tube in the posterior columns.[1] This view is in most perfect accordance with the result of Leyden's[2] examination of two cases of congenital internal hydromyelia. There could be no doubt that the condition in both cases was one of true hydromyelia, for both children (aged respectively two and a half and two years) suffered from congenital hydrencephalocele, which eventually caused death; and the cephalic tumour consisted of a hydrocephalic dilatation of the cerebellum, which, as is well known, leads, by implication of the fourth ventricle, to internal hydromyelia.

A review of the alterations found in the spinal cord in these two cases of real hydromyelia shows a peculiar analogy with those found in the syringomyelia of adults. With regard to this relation, Leyden lays stress on (1) the resemblance which a cavity extending along the cord from the lower extremity of the fourth ventricle downwards, and leaving the lumbar region unaffected, bears to the condition found in syringomyelia; (2) the position of the cavity in the posterior columns; (3) the fact that (as is also generally the case in syringomyelia), in the two cases mentioned, the canal was surrounded by a layer, more or less thick, of consistent tissue, which had in one case a horny and in the other a gelatinous character resembling that of the ground-substance of the spinal cord. This tissue developed itself around the canal, and in certain sections took on the character of a tumour. In certain portions of this mass appeared[3] (as in the previously mentioned cases of Simon and myself) a breaking down in the centre, so that a cavity appeared within the new growth, situated entirely in the posterior columns; (4) the layer which surrounded the canal appeared in the one case thin and tough, in the other soft but greatly developed, a difference found also in the cases of syringomyelia; (5) the cylindrical epithelium

[1] Compare Waldeyer, "Ueber die Entwicklungsgeschichte des Central-Kanals im Rückenmarke." Virchow, 'Arch.' 68, p. 20, 1876.

[2] Leyden, "Ueber Hydromyelus und Syringomyelie;" Virchow, 'Arch.' 68, p. 1, 1876.

[3] At least Leyden explains the fissure in this way.

lining the cavity (although there can be no doubt that it arose from the central canal) had in places disappeared, from which it follows that one can draw no conclusion from the absence of such epithelium in determining the nature of a cavity; (6) Leyden lays stress also on the relation between the affection of the posterior columns often found in syringomyelia (especially of the columns of Goll), and the condition of the same parts in cases of developmental irregularity; he considers the non-union of these columns in the median line as the starting-point of the affection.

Finally, Kahler and Pick have shown in a number of cases that considerable dilatation of the central canal, resulting simply from development and causing no pathological changes, may exist unobserved in the adult; also that, notwithstanding the existence of a normal central canal, a central cavity may have arisen from the enlargement of the central canal, and that, even in the case of cavities occurring in a situation unusual for the enlarged central canal (for instance in a posterior horn), one cannot deny the possibility that we have to do with diverticuli from the central canal.

After all this, there can hardly be a doubt that the view formerly adopted by Simon and myself,[1] and which seemed supported by the facts observed, must be given up, in that the syringomyelia can no longer be regarded as quite independent of changes in the central canal, but a certain relation to such changes must be acknowledged.

The following description of two cases will serve as a contribution to the solution of the question under discussion.

Case I.—Hohman, aged 67, a carpenter, came to the Charité, June 6, 1871; was treated in the department for nervous diseases from Jan. 10, 1872, till Feb. 1, 1873; from this time, till May 1874, was in one of the general wards; after which he was treated in the department for nervous diseases until his death, which took place May 24, 1874.

From the history taken in the general ward in 1871, it appears that the patient had suffered from intermittent fever, which he contracted at Wilhelmshafen a year before he came to the hospital, and of which he had had an attack three weeks

[1] Cf. Fr. Schulze, Virchow's 'Archiv,' vol. 87, p. 510.

before admission. He also experienced difficulty in breathing, and complained of a sensation as if inspiration were hindered beneath the manubrium sterni, this sensation following a persistent cough. He was described as a decrepid individual. Objectively, stridor, superficial breathing, enlargement of the spleen, and soon a great increase in the amount of urine, which had a low specific gravity (1006–1015). The maximal amount in 24 hours was 5400 cc., the minimal 2500 cc. Patient had an insatiable appetite. He did not know how long the polyuria had existed.

On June 30, 1871, it was noticed that the legs were swollen; patient felt as if he were encircled by an iron band. Examination showed no abnormality of sensation or localisation. The temperature, as far as noted, never rose above 37·2°, and averaged 36·5°.

Neither laryngoscopic nor other examination revealed the cause of the stridor. Substernal struma was suspected, but objectively only bronchial catarrh, with tough viscid sputum, could be established.

In August codein was administered, after which the appetite lessened, and the quantity of urine fell to 1600–2800. The appetite did not increase after the codein was discontinued (August 17). The urine gradually became more abundant, so that, for example, the quantity on Sept. 15th was 4900.

No sign of paralysis of lower extremities before Sept. 23rd, 1871. Two days before this date he had fever, which set in with a temperature of 39·8°, and on the evening of the 23rd reached 40·0°. On this day were noted pains in the lower extremities and inability to stand. On the next day the temperature was normal. No cause for the fever was found. After this, recurring headaches were noted, which lasted several days. The appetite had returned on the first of October. The polyuria remained unaltered.

On Nov. 4th, 1871, a careful examination was made, in which appear the following statements:—Marked tremor in lower extremities, and severe headache, the latter of which "rises from the small of the back." The patient cannot walk. In bed he can lift the legs with a zigzag movement to an angle of 30°. Pains in the legs. Sensibility in the lower

extremities seems absent, but examination shows that deep pricks of the pin are felt. The results are somewhat uncertain, on account of the inattention and lack of intelligence of the patient.

The lower extremities lie always extended, and it is nearly impossible to bend the knee; the movement being attended with great pain. Flexion at the hip-joint is possible, but difficult, and painful. The ankles are also stiff. Rotation is the only voluntary movement possible in the lower extremities. Patient says he is unable to stand. Even when placed on the edge of the bed the knees are not flexed, though one can with an effort bend the leg to a right angle; left to itself, it returns to the extended position. If one raises the whole limb in bed, and lets it go suddenly, it does not drop at once, but gradually (and sometimes by successive jerks), as if let down by the will of the patient. The cause is probably a stiffness in the muscles of the hip-joint. Patient is unable to stand; says that when he could still walk in the general ward, he noticed trembling in the limbs. On micturating, patient complains of feelings of pressure and constriction.

The quantity of urine on Jan. 11th, was 4900 cc.; Jan. 12th, 5600 cc. (specific gravity 1006); Jan. 13th, 5500 (specific gravity, 1000). On the 12th and 13th we found by Trommer's test a substance reducing cupric oxide; the polarisation apparatus showed 1·3 per cent. of sugar.

On the 16th, 6600 grammes of urine were passed, with a specific gravity of 1007, no precipitate. On the 18th, 6000 grammes (specific gravity 1007); a distinct precipitate by Trommer's test.

The notes upon the case after return to the general ward are confined to measurements of the urine, which were very variable, ranging from 1200 cc. to 5500 cc. The treatment consisted principally in injections of morphia, in the course of which an erysipelas was developed, with high fever. This complication was treated by injections of carbolic acid.

On the 7th of May, 1874, the patient was sent back to the clinic for nervous disease. Increased quantity of urine seems not to have been noticed recently, although occasional enuresis

rendered exact measurement of the urine impossible. Abnormal constituents no longer found in urine. The following notes were taken in the Clinic for nervous diseases on May 21, 1874.

Temperature.—Morning, 37·0°; evening, 37·2°.

Patient says he cannot lift his legs. Slight flexion only possible in knee-joint; somewhat more in ankle and toe-joints. On attempting to perform dorsal flexion of the foot, one experiences a resistance from the tendo Achillis which prevents the complete flexion. This resistance is still more marked on the right. No trembling in lower extremities. Limbs emaciated, as is the whole body. Faradic excitability of the muscles normal. Pricks of the pin in the lower extremities cause a grimace; other tests of sensibility impossible. The function of the upper extremities is affected; he can perform all movements, but with very little strength. No disturbance in articulation from affection of the tongue, and no atrophy of the same; no partial atrophy of skeletal muscles noticeable. Urine very dark, brown-red, without sediment, 1030; no albumen, no biliary colouring matter, no sugar.

Patient complains since yesterday of difficulty in deglutition, and eats only fluid food. Experiences a sensation as if he were "laced in" in the region of the manubrium sterni.

May 22, *Temperature.*—Morning, 36·5; evening, 36·3. The twenty-four hours' urine cannot be measured, because patient has passed the greater quantity in bed; there seems, however, to be no increase. If one raises the thigh, the leg drops by its own weight, and no stiffness appears in the knee-joint. Patient called attention to a swelling in the submaxillary region; and examination showed enlargement of the lymph glands. Mercury ointment and a Priessnitz compress ordered. The glandular swelling the next day decidedly increased, and filled the space between the ascending ramus of the jaw, and the cervical vertebræ, extending downwards to the angle of the jaw.

Marked collapse; hands cyanotic; radial pulse hardly perceptible, 78, sometimes intermitting. Patient died on the morning of the 24th.

Autopsy.—Exceedingly emaciated subject, nowhere œdema.

On both thighs swellings the size of walnuts, which on section prove to be hæmorrhages into the subcutaneous cellular tissue between skin and fascia. In the region of the angle of the lower jaw a swelling, shown by section to be a purulent infiltration of the enlarged parotid gland. Lower down, separate swollen glands. Swelling of follicles at the base of the tongue. Cartilages of the larynx partly calcified, mucous membrane of larynx normal. A stenosis in the upper part of the trachea, caused by projection of the cricoid and three first tracheal cartilages, narrowing the lumen of the trachea. On the outside of this prominence lies the lower end of the left lobe of the thyroid cartilage. Thyroid cartilage not hypertrophied, on section normal. In the œsophagus, somewhat below the tracheal stenosis, a pouch the size of a hazel-nut, with a neck of narrower diameter. *Lungs*, marked emphysema anteriorly, and chronic catarrh. Central softening of the right suprarenal capsule. *Kidneys*, hyperæmic. Mucous membrane of *stomach* thickened and reddened. Nutmeg *liver*. *Spleen* friable. *Bladder* contracted, containing a moderate quantity of urine; mucous membrane normal. Intestines and genitals not examined. Subpericardial fat tissue strongly developed. *Heart* large, flaccid, right ventricle greatly dilated; papillary muscles somewhat flattened. Heart walls rather thick, heart substance pale. Valves normal.

Spinal cord.—On opening the dura mater a small quantity of serous fluid escapes. The dura is everywhere smooth, nowhere adherent. The pia is not thickened in any part. The cord itself is in the upper part noticeably soft and glistening. On the surface of the cervical enlargement, the portion of the right posterior column limiting the median fissure shows a dirty-brown colour on a width of about two millimeters. This discoloration extends to about 29 cm. above the conus medullaris. Sections in the upper part of the cervical enlargement show an exceedingly glistening surface, above which projects a tumour of dirty-brown colour, measuring 4 mm. transversely, and 2–3 mm. longitudinally. The tumour lies in the region of the right posterior column, the surface of which remains free. It involves the grey substance, but appears not to reach the central canal. The same brown

mass appears on all sections made below this point, extending in some places beyond the median line. A section 4 cm. above the conus medullaris presents microscopically no abnormal appearance. The consistence of the cord is here good, while 14 cm. above the point of the conus medullaris, the cord is already softened, and shows the circumscribed red mass already described. Somewhat below this point in the lumbar enlargement both posterior columns present a brownish discoloration. The anterior columns are everywhere free, as are also the anterior horns. Nothing abnormal in the left lateral column; the right appears somewhat glistening and compressed, discoloured in spots. Nothing abnormal upon the roots or the cauda equina.

Cranium.—Dolichocephalus, somewhat asymmetrical, the right parietal bone being rather more prominent than the left. The diplöe on both sides of the sagittal suture, about 2 cm. posteriorly to the coronal suture, has almost disappeared. The sutures are externally well preserved, on the right strongly reddened. The frontal and both parietal bones are covered with red points and small prominences; on the right parietal bone appears one large and one small exostosis.

Brain.—The dura mater is strongly adherent at limited points in the frontal region. Pacchionian bodies along the longitudinal suture, corresponding to the thinning of the skull. The external surface of the dura mater shows nothing else abnormal. Fresh coagula in the longitudinal sinus. Inner surface of dura mater smooth, no pseudo-membraneous deposit.

Pia mater shows nowhere opacity nor thickening; its vessels are moderately filled; can be peeled off without loss of substance. Neither the vessels nor the nerves at the base of the brain present any abnormal appearance. The convolutions are rather small and pale; the grey substance of medium thickness; no infarctions in either the grey or white substance of the hemispheres. The white substance is moderately rich in blood. Nothing abnormal in the large ganglia. The right posterior column of the upper cervical cord shows the same discoloration as is seen in the cervical enlargement. One section appears, as above described, a dirty-yellow consistent

mass projecting from the cut surface in the region of the right posterior horn, and the inner part of the right posterior column. The columns of Goll in the medulla oblongata appear discoloured. At the apex of the calamus scriptorius the sections present no abnormal appearance, nor does the floor of the fourth ventricle; sections through the pons and peduncles normal.

Fresh microscopical examination of the apparent tumour in the upper part of the cervical region shows everywhere numerous brown-red pigment masses, which lie imbedded partly in coarsely fibrillated connective tissue. In places the ground-substance is not fibrous, but finely granular and streaked, resembling neuroglia. The pigment masses which, with a magnifying power of 250 diameters, appear round and remarkably similar in size, are partly arranged in rows, as if escaping from vessels. In a few places it is evident that they lie in or upon the walls of the vessels. In the great majority of the masses this arrangement does not appear, but they lie irregularly and in heaps on the ground-substance described. In the preparations made by teazing out with needles, the vessels do not appear notably increased.

Examination after the hardening of the cord shows a new-growth, extending from the upper part of the cervical to the lumbar enlargement, and occupying the posterior part of the cord. The lowest section on which it is visible lies 11 cm. above the apex of the conus medullaris, in the right posterior column. The situation of the new-growth varies greatly, and on some sections two separate masses appear; the variations in seat and form of the tumour are so great as to make it hardly possible, or at least very tiresome, to describe each separate section offering peculiarities, those which have been pictured sufficing to give a general idea of these variations.

One sees that the new-growth (Fig. 1, upper part of cervical enlargement) lies principally in the right posterior column, and here extends to the grey substance, but it encroaches also upon the left posterior column. Its posterior boundary is pointed, and extends, less markedly developed, to the periphery of the region of the column of Goll. In Fig. 2 the growth occupies equally the anterior parts of both posterior columns, has displaced the grey substance of both horns, and extends,

pointed, backward to the periphery. In Fig. 3 the posterior columns are included to a still greater extent, especially the right, and the commissure as well as the grey substance on the right is lost in the new growth. In Fig. 4 (about 27 cm. above the apex of the conus medullaris) two distinct masses are visible: one, of rounded form occupying the anterior part of both posterior columns; the other, situated on the inner boundary of the right posterior horn, long and narrow: in a section made somewhat deeper, the new-growth is situated no longer at the boundary of the posterior horn, but within the same, having taken its place. In Fig. 5 the mass in the right grey substance unites with that in the posterior column (here again principally in the right), thus causing a destruction of the grey substance except an outer strip; apart from this is seen a small mass of the growth in the left posterior horn. On deeper sections through the dorsal region, the growth gradually leaves the grey substance, leaving it at last quite free; and in the lumbar region there, separates itself again, a mass which lies at first in the anterior part of the posterior columns; it runs, pointed, backwards, ending at the periphery of the cord, and comes to lie more and more in the right posterior columns (Fig. 6), and at last, growing smaller, but preserving its form, it lies only in the middle and posterior part of the posterior columns, while the point no longer reaches the periphery.

Irregular as are its form and seat at its different heights, the tumour lies nowhere in the anterior regions of the cord, and particularly nowhere in the anterior or lateral columns; even in the sections in which it involves the anterior grey substance, the new-growth appears to have pushed forwards from behind.

Already with the naked eye one can distinguish an outer mass, coloured strongly red by carmine, and forming in places a broad and in places a narrow ring; and a delicately coloured transparent inner part. The latter appears to contain a split, partly simple and partly branching (the same is seen in the rounded mass in Fig. 4). This apparent split shows in many sections, even to the naked eye, peculiar zigzag lines. No real split, however, exists, and the appearance of one is only the result of the imperfect colouring in the inner parts, and in

some sections apparently the result of partial separation, due to mechanical influence. It is only in Fig. 6 that the new-growth appears to consist of a regular mass; and even here one sees with a higher power an irregular part, very narrow, and easily distinguishable by its transparency and delicate colouring. This part is broader several sections above Fig. 6, and more plainly marked.

The central canal is everywhere, excepting where the new-growth encroaches deeply upon the commissure, plainly visible as a central thread of ependyma without special growth in its neighbourhood. Wherever the new-growth reaches the anterior commissure, and involves the portions behind the commissure, the canal fails, and no sign of it is visible in the growth. The structure of the growth is very peculiar, complicated, and difficult to analyse. The bundles of connective tissue and the vessels form truly labyrinthic convolutions. My friend Professor von Recklinghausen (of Strassburg), to whom I had an opportunity of showing sections, had the kindness to communicate at my desire the result of his examination. This communication I here insert.

"The difficulty of explaining the sections is very great; the relations in the four sections given me are so various, that it is hard to bring them under one head. I have attempted again and again the explanation from the beginning, hoping to reach the true solution. The following is now my opinion:

"In all sections through the tumour appears a fibrous substance more or less rich in pigment, very poor in cells, with here and there rather larger cells, which represent perhaps ganglion cells altered in shape. This tissue stands in close connection with the neuroglia of the surrounding posterior columns, in which the atrophied nerve-fibres are recognisable, so that the tissue makes altogether the impression of being formed by the neuroglia, and resembles in texture an inflammatory product of the spinal cord. The peculiarity of the formation consists in two things: 1st, the serpentine canals, lying close together and partly in connection with each other; probably branches of blood-vessels distinguished by an extensive, very firm, and almost structureless layer of varying thickness, which follows the almost unaltered intima;

practically no trace of elastic tissue or unstriped muscular fibres appearing, so that it is impossible to decide whether we have to do with veins or arteries, probably, however, the latter; the lumen is very narrow, contains no distinct blood-corpuscles, and is perhaps at least in part obliterated. 2nd, the peculiar papilla-like, or better, shirtfrill-like bodies, which appear in two places in the sections. The body appearing in the section marked (x) forms a completely closed ring, whose centre is quite filled with delicately-fibred connective tissue. The ring of the second body is on one side not closed; here, rather, the delicately-fibred connective tissue filling the centre is continuous with the connective tissue imbedding the whole body. Also here throughout, no lumen, no fissure. Each ring is composed of feebly-striped, transparent tissue, apparently wanting in cells. This tissue is not stained by carmine, and resembles the tissue already described in the walls of the vessels, differing only in that its appearance is more glistening. I cannot but give it the name *sclerotic connective tissue*, although it lacks the firmness and strong glance of sclerotic connective tissue in other organs (skin, serous membranes). This sclerotic tissue makes in the most complicated form *folds*, not properly papillæ; on the tops of the folds, and often in the valleys between the folds, the superficial layer is delicately striated, so that here the impression is given of vertical section through layers of flat epithelium, but neither defined cells nor elastic tissue can be recognised. I recall similar forms, especially as regards the folds, only in the walls of canals; for instance, on the inner layer of arteries, whose entire walls have been by some process thickened. The same appearance may be artificially produced on cross-section, through vessels by folds in their walls. At first, having these thickened vessels in mind, the thought suggested itself to me that perhaps these appearances represented a very large vessel, with thickened walls and lumen filled with adhesive connective tissue. Still, the diameter of the closed ring is so great as hardly to be attained even by a dilated blood-vessel in the spinal cord. There remained then only the supposition that another sort of canal gave rise to this appearance: it was probably a syringomyelia, as supposed by yourself,

whose lumen had originally at least a diameter equal to the longest diameter of the present ring. Further, one must suppose that the inner layer of the wall surrounding the cavity took on a sclerotic character, while the outer wall, or, what is the same thing, the imbedding, almost adventitious connective tissue layer increased immensely in volume, so that the inner layer became folded, and the lumen narrowed. Finally then, the lumen must have disappeared through the formation of adhesive tissue, which afterwards in some places increased (also with breaking through of the sclerotic layer from outwards inwards, or in the opposite direction), and became continuous with the external basement substance. The situation of the pathological formation, together with the extent of the affection, seems to me to warrant the assumption that the whole formation represents a syringomyelia, with consecutive chronic inflammation thickening the walls and obliterating the lumen (periependymic sclerosis). The great difficulty lies in the unusual layer of sclerotic tissue in the wall (sclerotic, of course, in the common meaning of the word among pathological anatomists); still I remember to have seen preparations with Leyden, I think they were from his Case 3, 'Klinik der Rückenmarkskrankheiten," II. 454, in which such sclerotic tissue existed. I used at that time the term, but do not remember with certainty whether the tissue only existed on the vessels—it is certainly not rare for it to be found on them in other inflamed organs."

To assist in understanding the descriptions given of the microscopic appearances, I add a *figure* (7) which presents a portion of the new-growth lying near the right posterior horn (from Fig. 4, Hartnack, IV. oc. 2). One sees the papilla-like formation springing inwards from the strongly coloured tissue. Surrounding this formation, and pushing into its depressions, appears a layer-like tissue, not stained by carmine, but retaining the chromic-acid colouring. The central portion between the two irregular edges is filled with a finely striped, feebly-coloured tissue.

I add some details to this description, which fully coincides with the results of my own examination. On the sections in which the apex of the new-growth extends to the periphery

of the posterior columns (Figs. 1–3), large separate groups of nerve-tubes are surrounded by bundles of thick fibrillar connective tissue. The bundles of nerve-tubes, on the other hand, are penetrated by stripes of fibrillar tissue, which limit smaller groups of nerve-tubes in the bundles. In certain spots the groups of nerve-tubes thus limited present a normal appearance, the medullary sheath being plainly coloured by the potassic chromate; in other places the cross-section of the tube is decidedly lessened, and instead of the yellow medullary sheath, one sees the contents of the tube not coloured at all, or feebly red; in some sections no axis-cylinder can be made out in these small tubes; in other sections the axis-cylinders are plainly visible as little red points, much smaller than the normal axis-cylinders in the unaltered nerve-tubes of the posterior columns (Cf. Fig. 8). Over and between the separated nerve-tubes lie numberless cross-sections of blood-vessels, with adventitia enormously thickened and fibrillated. The bundles of nerve-tubes in Figs. 1 and 2, appear as small holes (*a*), the vessels as small red points Fig. 2 (*b*). The posterior columns themselves, wherever they are free from the growth, are unaltered. No grey degeneration appears in the columns downwards from Fig. 1. Above the commencement of the new-growth in the upper cervical region, on the other hand, the posterior columns appeared at the antopsy grey and transparent; these portions were, however, not available for microscopical examination. The grey substance of the anterior horn is anteriorly everywhere intact, except in those sections in which this substance is encroached upon by the new-growth. It is especially to be noticed that the ganglion cells, even with a low power, appear in normal number and form, and with plainly marked off-shoots. In the posterior part of the anterior horn, on the other hand, the grey substance, at the points of junction with the new-growth, is, by the loss of medullated nerve-fibres, more strongly coloured, of a spongy appearance, and presents a thick network of the finest fibres and spider cells, at the border of which tissue appear single ganglion cells, somewhat smaller, flatter, and containing less protoplasm than normal. The alteration in the grey substance is, however, every-

where limited to the immediate neighbourhood of the tumour.

Case II.—Stimmel, a carpenter, aged 58 years, admitted into the Clinic for nervous diseases of the Charité, August 28, 1875.

As the history of this case has already been published by my former assistant, Dr. Wernicke,[1] I shall not repeat it here, especially as the symptoms observed during life were not due to the lesion in the spinal cord, but to a tumour in the pons, and the publication was limited to the results of this tumour, while the case interests us here especially on account of the cavity in the *spinal cord*. Only the description of the tumour in the pons is therefore quoted here.

Autopsy: Brain.—From the floor of the fourth ventricle to the left of the median line projects a tumour-like elevation, 1½ cm. long and 2 cm. broad. This elevation appears to be formed by the junction of three smaller ones. Its posterior boundary lies nearly 1 cm. distant from the apex of the calamus scriptorius, its anterior boundary is 1 cm. distant from the corpora quadrigemina. The surface of the tumour is permeated with blood-vessels, varying in size and moderately filled with blood. The tumour projects nearly ½ cm. into the fourth ventricle, is quite firm to the touch, and grey-red in colour. No striæ acusticæ visible on either side. The tumour does not extend into the crus cerebelli ad pontem, but the left corpus restiforme is in its inner portion evidently filled with new growth. No sign of new growth found on section of the separated pedunculus cerebelli. A section through the middle of the tumour shows that it does not reach forwards as far as the anterior division of the pons; to the right it hardly passes the median line, and to the left it is limited by the lateral boundary of the pons. Section through the region of the crossing of the pyramidal tracts shows no evidence of new growth. No further pathological changes found in the brain beyond a moderate dilatation of the ventricles.

The dura mater of the *spinal cord* presents nothing peculiar; the veins on the posterior surface of the pia mater in the lower part of the cord are somewhat tortuous; the pia itself is transparent, not thickened, nowhere adherent to the dura. Sec-

[1] 'Arch. f. Psychiatr. u Nervenkrankh,' vol. vii. p. 513.

tions through the cervical region just below the crossing of the pyramidal tracts show a grey discoloration of the portions of the posterior columns bounding the posterior septum. On sections through the cervical enlargement appears a soft, grey-red mass, occupying the whole anterior part of the posterior columns, which mass reaches laterally to the posterior horns, pressing them somewhat outwards, and anteriorly extends without defined boundary into the commissure. The posterior horns are still plainly recognisable, also the anterior horns, although parts of the latter seem involved in the new growth. The posterior portions of the posterior columns appear free from new growth, but a brown discoloration extends along the septum. The tumour described is plainly visible in the lower cervical, but ceases at the commencement of the dorsal region, sections below being those of an apparently normal cord. The consistence of the spinal cord in the regions occupied by the tumour is soft, in form flat, and elsewhere its form and consistence are normal. The posterior roots present no abnormal appearance.

Microscopical Examination.—No sign of new-growth appears in the cervical region; above the cervical enlargement, on the other hand, the middle portions of the posterior columns show grey degeneration, as is seen in the illustration, and the anterior boundary of the degenerated portion being at the commissure. In the cervical enlargement (Fig. B) appears a cavity situated in the posterior columns, surrounded by a ring of varying breadth, coloured by carmine. This ring is lined by a narrow zone of tissue which has not taken the staining. The remaining portions of the columns of Goll are degenerated. The seat of the cavity in the following sections, as well as the breadth and form of the ring surrounding it, may be seen in Figures C, D, E; one sees that in Figure E the cavity comes to lie in the right grey substance, and a new mass appears in the right posterior columns. This mass is of the nature of a new growth, and shows a delicate colouring at its edges. The cavity ended in the dorsal region; its termination could not, however, be followed, as the part of the cord in which it ended was not

available for examination. The central canal (with cylindrical epithelium) in Fig. A, appears somewhat widened, and in certain sections in this region, is filled partly with red blood-corpuscles, and partly with a homogeneous, clear red-coloured mass, as if coagulated, and containing single blood-corpuscles. Elsewhere the central canal lies in its normal position as a central ependymal thread, excepting in the sections where the circle of new-growth (as in Fig. E) extend far into the commissure so as to destroy the integrity of the latter. The ring of new-growth consists of an entanglement of fibrillar connective tissue, in which numberless nuclei are seen after staining with hæmatoxylin. On some preparations the ring appears in spots to be formed from distinct layers in the process of preparation. The outer part of the ring (in which the large number of cross-sections of vessels is noticeable) runs into the surrounding tissue in a zigzag manner, or with a network. The adventitia of the vessels is everywhere remarkably thickened. Here and there at the periphery of the ring, and in the grey substance in its neighbourhood, can be seen masses of dark-brown pigment, and minute fresh hæmorrhages (collections of red blood-corpuscles). In Figs. A and B, the inner border of the ring consists of a narrow strip of connective tissue with parallel fibres, coloured feebly or not at all by carmine, but well by hæmatoxylin, which brings out also numberless nuclei; on other sections no such inner boundary can be demonstrated. Of special interest is the fact that on several sections—for example, that represented in Fig. B—appear two bodies, not very far apart, directly under (outside) the inner rim which is not coloured by the carmine. These bodies resemble closely the body near the right posterior horn in Fig. 4 (Case I.), the microscopical appearance of which is shown in Fig. 7. There appears in them the same shirtfrill-like folds, with the same corresponding layer-like tissue as in the former place, only that the central finely-striped, delicately red-coloured tissue fails. The boundary line of the bodies towards the lining membrane, under which it laid, was partly simple, and in part appeared studded with similar papilla-like projections and depressions, similar to those in the inner side of the body itself, surrounded in like manner by the layer *a*.

As we have seen, the first case is one of *new-growth*, the second one of *cavity-formation*. If we notice, however, the situation of the new-growth in the first case, its form and its extension through the spinal cord, the idea involuntarily presents itself, that the case of new-growth is allied to that of cavity-formation. Its seat in the posterior columns and posterior horns correspond exactly to that of the cavity described; the form of the new-growth has also, especially in the cervical enlargement, a noticeable resemblance to the form of the cavity which one finds in this region in cases of syringomyelia. The fact also that the growth in the one case, as the cavity in the other, ends in the lumbar region, shows a correspondence with most cases of syringomyelia. To this is added the peculiar formation of the tumour, which in many sections shows inside a particular body. All these facts make it highly probable that this body originated in a cavity, which originally has been a diverticulum of the central canal, as we have become acquainted with above, and that the tumour became gradually filled with the "sclerotic" tissue. The surrounding ring of connective tissue, strongly coloured by carmine, represents the ring which one commonly finds around a syringomyelia. We can only surmise the exact mode of occurrence of these changes (compare the remarks of Recklinghausen above).

The relationship of the new-growth in the one case, with the cavity in the other, is finally shown by the discovery of bodies, as above described, in the surrounding ring of the second case, closely resembling those described in the first case connected with the tough (sclerotic) tissue forming the inner border of the ring.[1] One can imagine that here the filling up of the cavity was interrupted, and that the shirtfrill-like folds had only

[1] This article was already written, when Dr. Pick showed me some preparations of Case III. in his work mentioned above (Kahler and Pick, 'Beiträge für Pathologie und patholog. Anatomie,' &c., Leipzig, 1879, p. 113), in which were present completely analogous, indented formations, in this case, however, starting from the inner border of a ring bounding a central cavity. They protruded into the lumen of this cavity; in some places there were remains of epithelium on and between the indentations. Cf. the interesting paper of Fr. Schulze quoted above, and a case related by Strümpell, 'Archiv für Psychiatrie und Nervenkrankheiten,' vol. x., p. 704.

become developed in separate spots, while in general the inner sclerotic rim laid smoothly on the outer ring.

There can hardly be any doubt that the changes in the spinal cord were congenital, and one must therefore assume that disturbance of development of the central canal ensued with cavity formation, accompanied by a tendency to development of fibrous new-growth. This development is often considerable in the surroundings of the cavity, and characterised by great vascular development and pigmentary deposit. The inner layers of the wall of new-growth take on meanwhile the peculiar sclerotic character described. In other cases there seems to be a tendency to the development of another form of new-growth (glioma, myxoma). Hæmorrhages often appear to accompany these processes.

The fact that in the second case a tumour was found in the pons, as well as a cavity in the spinal cord, might perhaps tempt one to conclude that a tendency to tumour-formation existed at the same time in different parts of the central nervous system. Were, however, the opinion correct which was formed from the histological examination, that in this case we had to do with a solitary tubercle of the pons, the idea of such a tendency must be given up. To-day the question could be definitely settled by the presence or absence of bacteria.

No symptoms appeared in the first case which could be attributed to the spinal-cord affection; the syringomyelia then ran its course here, as already often observed, quite without symptoms. The patient in the first case came to the hospital on account of his difficulty in respiration, and the discovery of the symptoms of sensation and motion was rather accidental, considering the mental condition of the patient. To sum up these disturbances, we find, paresis of the lower extremities, with appearance of stiffness; weakness of upper extremities, and a diminution of sensibility, whose boundaries could not be accurately determined on account of the patient's mental condition. There was no musclular atrophy, properly so-called (degenerative atrophy); the wasting of the subcutaneous tissue and muscles was general.

A symptom of especial interest was the increased quantity of urine and the occasional occurrence of sugar, which

one might well be tempted to connect with the alterations in the spinal cord; for, according to the observations of H. Dickinson,[1] dilatation of the central canal in the dorsal and lumbar region, together with proliferation of the lining epithelium, are noticeable, though not constant, in the post-mortem appearances of diabetes mellitus.

[1] Compare Senator, "Diabetes Mellitus und Insipidus;" Ziemssen's 'Handbuch,' xiii. 2, s. 139.

EXPLANATION OF THE ILLUSTRATIONS.

Fig. 1–6.—Cross-sections through the spinal cord of the first case (cervical and dorsal regions). Low power. (The details are as seen with Hartnack II.)

Fig. 7.—A part of the body (*c*) in Fig. 4, seen with Hartnack IV., ocular 2, tube drawn out.

Fig. 8.—Nerve-tubes surrounded by the fibrillary tissue of the new growth and deprived of their medullary sheaths, the axis-cylinder visible in many as a point. This view represents the point (*a*) in Fig. 1. (Hartnack VII.), ocular 3, tube drawn out.

Fig. A.–E.—Cross-sections through the spinal cord of the second case (cervical and dorsal regions.

Cc. The somewhat widened central canal.

a. Inner border of the ring.

b. Hæmorrhages.

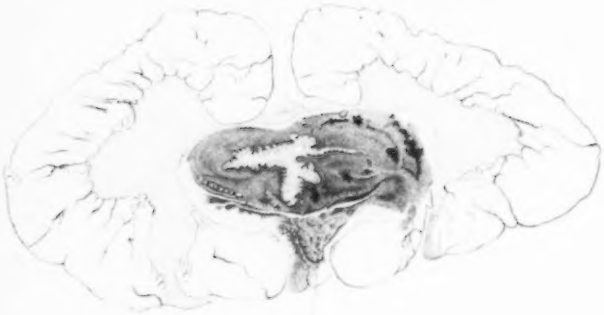
[Translated by D. L. Walton, M.D., *Boston*, U.S.A.]

Fig. 1.

Fig. 4.

Rob. Schwann del.

Fig. 2.



b

Fig. 3.

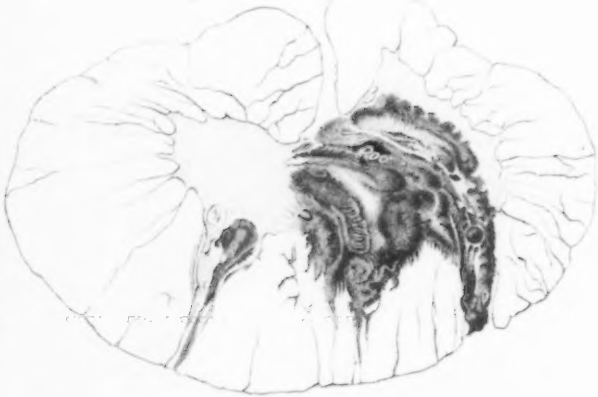


Fig. 3.

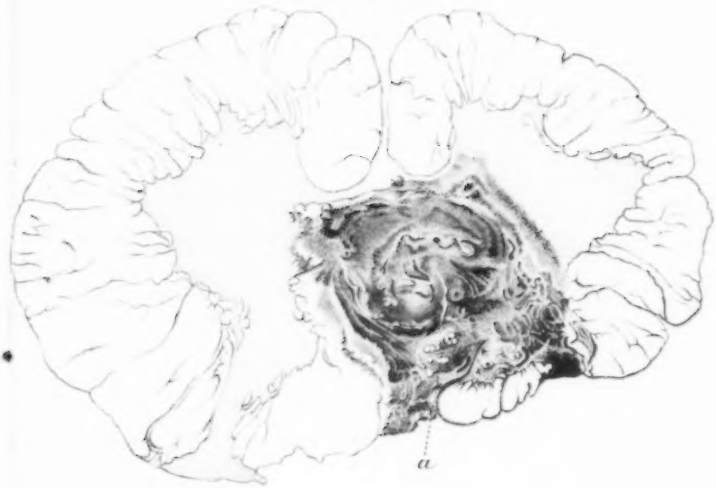


Fig. 6

C. Lane lith.

Fig. 1.

Fig. C.

Rob. Schmann del

Fig. B.

Fig. S.

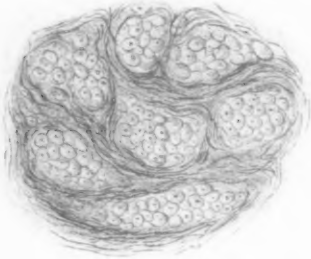


Fig. D.

Fig. A.

Fig. E

C.Lane lith.

CLINICAL REMARKS ON TUMOURS INVOLVING THE PARTS IN THE NEIGHBOURHOOD OF THE THIRD AND FOURTH VENTRICLES AND THE AQUEDUCT OF SYLVIUS.

BY J. S. BRISTOWE, M.D., F.R.S.

THE cases which I am about to narrate and discuss were cases which interested me much during life; and are all the more instructive that they each became elucidated in the course of time by a post-mortem examination. The first case was one in which a tubercular mass had destroyed the corpora quadrigemina, and by pressure displaced the iter downwards and forwards; the second was one in which a tumour sprang apparently from the valve of Vieussens and processus e cerebello ad testes, and had invaded the corpora quadrigemina and cerebellum, and become adherent to the floor of the fourth ventricle; and the third was one in which a tubercular growth occupied the third ventricle and implicated the adjoining optic thalami. The fourth and fifth cases scarcely belong to the same category; inasmuch as the fourth was a case of tubercle of the cerebellum, and the fifth a case of chronic hydrocephalus. But I have ventured to add them to the others because, in the former the cerebellar tumour pressed upon the floor of the fourth ventricle, to which fact probably some of the symptoms attending it were due; and in the latter, owing to obliteration of the iter, the fourth ventricle formed an independent cyst, and thus its most interesting pathological feature was the presence of disease in the vicinity of the lesions presented by the other cases.

The first case was that of a little boy of seven, whose illness terminated fatally after six months' duration. The earliest symptom of his malady was, it was said, loss of power in the right arm and leg coming on suddenly. But before long all

his limbs became paretic and tremulous on movement; and his head and neck also displayed tremors when unsupported. Further, he showed marked symptoms of ophthalmoplegia externa: protrusion of the eyeballs, ptosis, and paralysis probably of all the ocular muscles, though mainly of the superior and internal recti. Moreover, the eyes had a tendency to act independently of one another. Sight, however, remained perfect; the pupils (though at times unequal) acted to light and to accommodation; and there was no optic neuritis. His speech was drawling, and attended with tremors of the lips. There was absence of headache, vomiting and convulsions. Among subordinate symptoms, either present from the beginning, or coming on in the course of the case, were drowsiness, indistinct facial paralysis, exaggeration of the knee-jerk in both legs, with stiffness of the right, and loss of control over the evacuations. During the last few days of life acute symptoms came on, with considerable rise of temperature, which was due to the supervention of tubercular meningitis.

Now it was not difficult, during this patient's life, to make a shrewd guess as to the seat of his intra-cranial lesion, and even as to its nature. A young child is much more likely to suffer from a tumour than from sclerosis; and a tumour is, in such a case, much more likely to be tubercular than of any other character. That the child was suffering from a tubercular growth, was rendered almost certain later in his career, when symptoms of meningitis were added to his other symptoms. At any rate it was assumed when he first came under observation that, notwithstanding the absence of headache, vomiting, optic neuritis, and fits, he was suffering from cerebral tumour. The seat of the tumour was indicated by the ocular paralyses. It was evident that the nerve-trunks were not involved, and that the nerve-nuclei were not destroyed; and hence, either that the tumour was higher up or more anterior than the nuclei, or that it simply compressed them. The partial, and to some extent variable, facial palsy, with the absence of implication of any of the cerebral motor nerves below, and of any of the nerves of special sense, further seemed to indicate that the disease was in the corpora quadrigemina or their immediate neighbourhood. The presence of universal muscular

weakness, with tremors, and the absence of actual paralysis of the limbs, indicated rather symmetrical compression and irritation of the motor tracts, than their actual destruction by a tumour.

At the post-mortem examination there was found, in addition to recent tubercular meningitis, a solitary tubercular mass, about as large as a marble, imbedded in the corpora quadrigemina, destroying these bodies, and displacing and indenting the tracts of nervous matter situated to the outer side of, and below, the aqueduct of Sylvius.

The second case was in many respects much more interesting than the first. It was that of a young lady of twenty, whose illness extended over rather more than two years; and whose condition, up to two months before her death, was attributed to aggravated hysteria. The first symptom noticed was loss of power in the middle finger of her left hand, which interfered with her playing on the piano. To this succeeded deafness of the left ear, and dimness of the left eye; which before long also presented an inward squint. Later, she complained of numbness in the left arm and leg; and of some stiffness, associated with tremors, in the legs. The phenomena above described became slowly developed, one after another, during a period of eighteen months. During the next four months, however, the progress of her disease was rapid. She became deaf and blind; the left eye turned outwards instead of in; she spoke in a drawling, whining tone, and was at times incoherent; and her limbs became generally very weak, and all her movements were attended with tremors. On admission, two months before her death, she was for the most part in the condition just described. She could stand and walk when supported; but all her limbs were weak, and these and her head and neck trembled, as in disseminated sclerosis; she was quite deaf and blind; there was no ptosis, but the eyes diverged and acted at times independently of one another. The paralysis was most complete in the right external rectus; but apparently all the muscles of the right eye were weak. There was probably also some weakness of some of the muscles of the left eye, which also presented rotatory nystagmus. The pupils were unequal; and there was double optic neuritis. She

drawled in speech; and was affectionate, not to say amorous in her manner. There was slight paralysis of the right facial. When she had been in hospital about a fortnight, a mattery discharge took place from her right ear, which continued until her death. In this case, as in the last, the patient was very drowsy; and there was (at any rate to within a day or two of her death) no headache, sickness, or convulsions. During the the last few days of life, the weakness in her legs increased; she had violent general tremors at times; she presented difficulty of swallowing; the veins of the inner halves of the eyelids became swollen; and her temperature rose.

In this case, as in the former one, it seemed clear during life that the patient was suffering from a cerebral tumour. And the association of absolute deafness, with the ocular troubles, partial facial paralysis, and general paresis and trembling, rendered it pretty clear that the tumour was seated somewhere about the fourth ventricle, and was of considerable extent. There were some points in the case, however, that added a little obscurity to it. Was the loss of power, limited to a single finger (which was her earliest complaint), referable to the tumour, which, as it grew, ultimately caused her other symptoms? Could the persistent, though lately developed, otorrhœa have any relation to a tumour occupying the substance of the brain, or was there a mere chance association between them?

The post-mortem examination showed that the diagnosis made during life was accurate; and suggested an explanation of some of the other incidents of the case. A largish growth was found springing apparently from the valve of Vieussens, and the processus e cerebello ad testes, which was adherent to the floor of the fourth ventricle in nearly its whole extent, and had involved the testes and cerebellum. But it was found also that the dura mater at the base of the skull, in every one of the fossæ, was the seat, in a greater or less degree, of the same kind of disease. The discharge from the ear was doubtless dependent on the latter complication. I could not, however, determine that any of the paralytic phenomena were due to involvement of cranial nerves, or narrowing of their foramina of exit.

The third case was that of a little boy, four years old, and was obviously (as will be seen from a perusal of the history) one of tubercular growth in the brain. The symptoms of his cerebral affection lasted nine months. They began with tremors, and slight loss of power in the left arm and leg; which were followed in the course of a month or six weeks by tremors in the limbs of the opposite side. These tremors were like those of disseminated sclerosis. The left side of the face also presented spasmodic movements at this time. About six months from the beginning of his illness (the symptoms having made gradual progress during this time), the left arm and leg had become rigid; the tremors of the right arm and leg had increased; there was some twitching in the left side of the face, with paresis of the left facial nerve; he had commencing double optic neuritis; he drawled in his speech; he was extremely drowsy; and he passed his evacutions into the bed. These symptoms presented little change, up to a week before his death; when his temperature rose, he vomited, and presented other symptoms of acute meningitis, of which he died. In this case, as in the others, there were no fits, no headache, no sickness, and the appetite was extremely good. It is important also to note that there was no paralysis of any cerebral nerve. There was, no doubt, weakness of the left facial, but this was clearly hemiplegic.

Where was the tumour that caused the symptoms above described? I confess I thought it would be found in the cerebellum. And I still think that this diagnosis was justifiable. The tumour, however, was found occupying the third ventricle, and involving to some extent both optic thalami. And I am inclined to think that, with the guidance of such cases as the first two narrated, a correct diagnosis might have been arrived at.

In reviewing these cases collectively, it is impossible not to see that they presented many features in common, which were probably due to the fact that the same tract or tracts were involved, and the same tract or tracts avoided, in each; and, at the same time, many points of distinction which were clearly referable to the additional involvement in each case of special centres or regions.

The chief points of resemblance between them were the following:—general paresis, with tremors like those of disseminated sclerosis; drawling speech, reminding one somewhat of that observed in myxœdema, and in one case attended with marked tremors of the lips, and thus presenting a close resemblance to the speech of general paralysis; remarkable drowsiness, with more or less emotional disturbance and loss of control over the emunctories; and in association therewith absence of anæsthesia, headache, convulsions and sickness, and the presence of an excellent, if not voracious, appetite.

The chief distinctive features, which may be attributed to difference of seat of lesion, were the following. In the first case, paralysis of both third nerves, and probably of both fourth nerves, without implication of the irides or muscles of accommodation; due probably to compression of the nuclei of origin of these nerves. In the second case, deafness on both sides, with some involvement of one, if not of both, third nerves; due probably to extension of disease into the auditory nuclei, and to pressure on the nuclei of the third nerves. In the third case, the absence of all specific phenomena; owing to the fact that the tumour was seated above the origins of the motor cranial nerves.

There were other phenomena presented by the cases, to which more or less importance may be attached; but to which, on account of their frequency in brain affections, I have not ventured to assign specific value. Among them were, paralysis of the sixth nerve, observed in one, if not in two, of the cases; incomplete facial palsy, probably independent of direct involvement of the seventh nerve; optic neuritis, which was marked in two of the cases; blindness, probably the consequence of neuritis; and the symptoms due to the supervention of meningitis observed shortly before death in the two tubercular cases.

The fourth case presented a close superficial resemblance to the third. The patient was four years old; his illness lasted for nine months; he had in the beginning, and for some months subsequently, tremors of both arms, and of the head and neck, which (so far as the arms were concerned) gave way to rigidity some time before death; his speech was drawling and monotonous; and he had double optic neuritis with blind-

ness. Further, he never suffered from sickness. But he suffered constantly from what none of the other patients had experienced, namely headache and epileptiform seizures; he presented, more markedly than any of them, the so-called "cerebellar gait;" and, in the course of his illness, paralysis of the left sixth and seventh nerves came on. Pathologically the case was most like the second; for although the tumour (which was of somewhat larger size) was a cerebellar tumour, it exerted manifest pressure on the floor of the fourth ventricle, mainly its left half. It was doubtless owing to this pressure that the patient had paralysis of the left sixth and seventh nerves; and probably owing to the same cause that he suffered from tremors of the head and neck and arms, and that his speech was drawling.

The appearances discovered after death showed that, in the fifth case, the lesion from which the patient suffered was of old date. It consisted, in fact, of dropsical effusion into the lateral and third ventricles, into the fourth ventricle, and into the central canal of the spinal cord. An interesting point, however, in the case was that the dropsical accumulation in the fourth ventricle was cut off from the accumulations in the neighbouring cavities.

The symptoms (excepting that the head was unusually large) did not point to the nature of the patient's malady. He died of a convulsive attack, of which he had had several during the last four or five months of his life. And his other chief symptoms were, feebleness and unsteadiness of gait, rigidity of the neck, headache, occasional double vision, and slight feebleness of mind with tendency to be noisy at night. He had no paralysis and no tremors.

CASE I.—*Tubercular Tumour of the corpora quadrigemina, followed by tubercular meningitis.—Paralysis of both third nerves, with ptosis, and tremors of head and neck, arms and legs.—No fits.—Sickness and headache absent until the last.—No optic neuritis.—Death.—Autopsy.*

Edward B., a schoolboy aged 7, was admitted under my care on the 2nd of August, 1882.

His illness began about the latter end of May. He is said to have gone to bed well, and to have got up the next morning with inability to walk properly, and with marked weakness in the right arm and leg. About a week later his eyes became affected, and gradually assumed their present condition. His speech became impaired at the same time. He has had no headache, no vomiting, no fits; his memory has not suffered, and his temper has remained good; his appetite has been good, his bowels constipated. He has been drowsy latterly, however; and for the last few days has passed his evacuations into the bed. He has also been getting weak and thin; and tremulous movements of the limbs have been gradually developing.

The child is thin, and has a large head; which, when he sits up, is usually thrown somewhat backwards. He is drowsy and dull; but when fully roused appears to be perfectly sensible. The limbs are all weak; but there is no distinct paralysis of any of them. When he sits up, the head trembles; when he moves his arms, the movements are attended with coarse undulatory tremors; and when he stands or walks, similar tremors affect the legs, and become transmitted to the body generally. The eyes are both unduly prominent; there is ptosis on both sides; and also distinct paralysis of the superior and internal rectus of the right eye, which looks outwards, and apparently a less degree of the same paralysis on the left side. The right pupil is slightly larger than the left; but both pupils act to light and accommodation. Sight appears to be perfect. The mouth is drawn a little to the right when the child laughs, but there is no other evidence of facial paralysis. The tongue is protruded straight. Speech is slow, drawling, and monotonous. No difficulty in swallowing. Hearing unaffected. No impairment of sensation. Superficial and tendon reflexes normal. Tongue clean; appetite good; bowels confined. Urine 1030; lithates, no albumen. No sign of thoracic or abdominal disease.

For the next three weeks there was no marked change in his condition. The description above given of him was in the main confirmed. Drowsiness and apathy varied in degree, but were generally present; he had no headache, or sickness; his evacuations were sometimes restrained, but occasionally escaped

unwittingly; the tremors continued, but it was noticed also that the lips trembled when he spoke; the paralytic condition of the eye-muscles remained much as it was; and it was ascertained that fundus of the eyes were healthy. It was noticed, further, that the occasionally sudden spasmodic tremors convulsed his whole frame. But there was no apparent loss of consciousness in these attacks. He retained his good-humour.

About the 25th of the month, and for a day or two afterwards, he complained of some occipital headache.

During the next month, again, there was very little change on the whole.—except, perhaps, that drowsiness and disinclination to speak or to take notice of things increased; that he became irritable, and apt to cry out at times, and even to try to bite when annoyed; that his evacuations were now all passed involuntarily; that he got less and less able to support himself in the sitting posture; and that his tendon reflexes became exaggerated. The tendency to retract the head continued; the tremors of his head and neck and limbs did not diminish, possibly increased; the slight paralysis of the right facial nerve was still observable; and the prominence of his eyeballs continued. On the 26th of September his eyes were examined by Mr. Nettleship, who reported as follows. "All the movements of the eyes are more or less imperfect, unless it be the movement downwards, which is better executed than the rest. The defect is greatest in the right eye, both as to the levator palpebræ, the superior rectus and the internal rectus. The paresis of the external recti is considerably less marked than that of the superior and internal recti. The right eye is directed outwards, and stands on a rather lower level than the left. The pupils are equal, of natural size, and act well to light. Action to accommodation I could not determine. The oculo-motor symptoms point to disease higher up than the trunks of the nerves, and missing the centres for the iris and ciliary muscles." Sight seemed to be perfect, and there was no optic neuritis.

For the rest of September, and during the whole of October, the condition of the child scarcely presented any change. There was no increase of paralysis; he still seemed to under-

stand, but was unable or indisposed to reply, and rarely spoke. But his attention could be aroused, and he could be made to laugh. The eyes were examined on several occasions, and the fundus was always found normal. He never had any distinct indication of headache, and never any sickness, or any fits.

About the 10th of November, it was noticed that he was a good deal weaker; that he had ceased to know his mother when she came; but that nevertheless he could see, and if a piece of cake was held up before him would grab at it; that the right pupil was much larger the left, and that both acted badly to light, and that there were no ophthalmological changes; that the mouth twitched a little to the left; that the patellar tendon reflexes were brisk, and that there was a tendency to rigidity of the right leg.

On the 17th it was remarked, that the boy had been getting more and more drowsy and indisposed to speak, though now and then inclined to be noisy; that the paralytic condition of the eyes remained, and that the ptosis was becoming more marked, especially on right side; that the tremulous movements had largely diminished, and that he still had no sickness, no obvious pain in head, and no fits.

On the 18th he vomited; and again he vomited on the 20th. The vomiting still continued; on the 22nd it was observed that he often put his hands to his head as if in pain, and that the tremors in arms and head had returned. After this he became weaker, and quieter, refused his food and continued to vomit occasionally; and his temperature, which had nearly always been normal, rose. On the 25th it reached 100; on the 26th, 101·2; on the 27th, 101; on the 28th, 101·6; on the 29th, 102; and on the 30th (the day of his death) it rose to 103·8. He died comatose, and without convulsions. The condition of his retinæ was not ascertained subsequently to the 11th of the month.

Autopsy.—The convolutions of the brain were flattened; the surface was dry and slightly congested, and the veins were engorged. The membranes at the base were thickened, and lymph was present in the subarachnoid tissue about the inter-peduncular space. There were a few miliary tubercles at the base of the brain; which were most abundant in the Sylvian

fissures. In the right fissure, just by the anterior convolution of the island of Reil, the tubercles were aggregated into a small tumour. The corpora quadrigemina, and apparently these alone, were occupied by a globular cheesy mass, about as large as an ordinary marble. This lay between the upper surface of the bodies and the iter, and was invested above and below by a thin remnant of the normal textures. The position of the corpora quadrigemina, as seen from above, was occupied by a smooth rounded expansion, which was due to the presence of the tumour and the consequent effacement of the proper contours of the part. The lower part of the tumour was received into a cup-like depression of the structures below the level of the iter; the latter passage being obliterated by pressure and forming from before backwards the segment of the circumference of a circle, with the convexity facing downwards and forwards. The substance of the brain was generally healthy; but the lateral ventricles were distended to some extent with fluid.

With the exception that the pleuræ presented adhesions that the lungs were thinly studded with grey tubercles, and that numerous larger yellow tubercles were scattered through the spleen, the abdominal and thoracic viscera were healthy.

CASE II.—*Tumour of valve of Vieussens, involving cerebellum and corpora quadrigemina, and adherent to floor of fourth ventricle.—Similar growth in dura mater of base.—Blindness, optic neuritis, paralysis of ocular muscles, deafness, tremors of head and limbs, and loss of power in limbs.—Mental affection—Discharge from right ear.—Death.—Autopsy.*

Lily M., a girl aged 20, was admitted under my care on the 4th of April, 1882.

Two years ago the third finger of her left hand became weak (not flexed), so as to interfere seriously with pianoforte playing, of which she was very fond, and in which she was clever. Later, she complained of dimness of the left eye, and deafness of the left ear, with sounds in it as of bells ringing. These symptoms persisted, and at the end of a year she began to see double. Five months later (seven months ago) she

presented a manifest inward squint of the left eye. About this time, too, she began to experience difficulty in walking; there was some tremor in the legs, they occasionally crossed one another when she attempted to walk, and she had to grasp at things for support; and she complained of numbness in the left arm and leg. She also suffered from fatigue, required assistance in dressing, and looked pale and ill.

From Nov. 20 to Feb. 8, she was away from home; and there is no record of her symptoms, beyond the fact that she gradually got worse. When she returned, the arms, and head and neck, were tremulous as well as the legs; and the left eye turned outwards. Her sight and hearing also were more defective than they had been; she was so weak on her legs that she could not walk without assistance; her speech was affected; and she talked at times incoherently.

From this time she rapidly got worse. Early in February the catamenia ceased; about the same time she complained that she was absolutely blind and deaf; and her talk was at times nonsensical and incoherent. She spoke in a drawling, whining tone; and talked constantly of herself, and in complaint of her blindness and deafness. She seemed not to recognise people, and was apt to give them new names, by which she continued to know them. She has generally recognised her mother, however. Her mother believes that she sees and hears, at any rate at times. Since January she has occasionally passed her evacuations in bed.

She is naturally of a gentle and affectionate disposition; and, excepting when (on one or two occasions) she has given way to temper, she has retained these qualities throughout her illness. She has never shown symptoms of hysteria, unless any of the above phenomena are to be regarded as hysterical; which was the view of those who sent her to the hospital.

The patient is a fair, well-nourished girl. She can walk with assistance in a shuffling way; but for the most part keeps her bed. She is blind and deaf, and speaks in a slow drawling manner. She has marked tremors of the arms (like those of disseminated sclerosis) when she uses them, and of the head and neck when she sits or stands. The legs also tremble to some extent. There is no loss of feeling; indeed perhaps a

little hyperæsthesia. The pupils are unequal, the left being larger than the right, and do not react to light. The eyes diverge; this appears to be due to an external squint of the right eye, the movements of which seem generally restricted. But it is difficult to be sure which ocular muscles are affected, inasmuch as the patient cannot see, and cannot hear so as to do what she is told. There is slight rotatory nystagmus of the left eye. Double optic neuritis is present: much swelling extending some distance from the disc, very tortuous and dilated veins, and several striated hæmorrhages. There is slight facial paralysis on the right side. She is constantly talking in a slow whining fashion, mostly on matters that concern herself, and often asks questions and to be kissed. If not gratified, she turns crossly on her right side. She does not try to do anything for herself; but if food be placed in her hand, she then puts it into her mouth. The legs and arms are all weak, but there is no definite paralysis. The patellar[1] and plantar reflexes are absent, and there is no ankle-clonus.

No affection of thoracic or abdominal viscera. Tongue clean. Appetite fair. Bowels confined. Urine pale, acid, no albumen. Temperature 99·2.

During the next fortnight her symptoms accorded in the main with the above description. She has occasionally got up; but generally lay in bed on her right side. She was very drowsy, and at times difficult to rouse. When awake, she was sometimes noisy, and called out for her mother. She seemed to know that she was left among strangers, but evidently did not know where she was. She often spoke of her condition of blindness and deafness, and of herself as the "mad Miss M." She evidently could not see or hear, as a general rule; but the sister and nurses believed that she could both see and hear imperfectly at times. It is certain, however, that her sense of touch was very acute; and any slight disturbance of the bedclothes or bed, made her move or stretch her arms in its direction. If she caught hold of any one's hand, she would grasp it and fondle it with both her hands, and press it to her bosom or carry it to her lips, and ask to be kissed. At

[1] This is the effect of the only record as to this matter. I believe, however, the tendon reflexes were examined on subsequent occasions and found present.

the same time her face assumed an extremely affectionate, if not erotic, expression. She generally fed herself greedily when food was put into her hands. The tremors and the paralytic condition of the eyes remained unchanged. She never passed her evacuations into the bed. On the 19th a discharge took place for the first time from the right ear.

During the next month the symptoms, on the whole, remained much the same as they had been. She was still often drowsy; but at times was restless; on the whole, however, much quieter than she had been. She still talked somewhat incoherently at times, and in her slow plaintive manner; and evidently did not always know where she was, or who the persons were about her. She was still blind and deaf, and the movements of the eyes were more or less independent of one another. She remained acutely sensitive, however, and understood many things. Thus, she was still inclined to be affectionate, she still fed herself when food was put into her hands, and if she felt stockings being put on her feet, she knew she was to get up, and she assisted in the process. The tremulous condition of head and neck and arms, and in a less degree of the legs, continued. The discharge from her ear persisted. No further paralysis had been developed; but the weakness of the right seventh, which had been before observed, was still present. Her tongue was clean, her appetite good; she had no sickness; her bowels were regular; she passed her evacuations consciously; her temperature was normal; and she was habitually got out of bed and placed in an armchair some time or other during the day.

On the 21st of May her temperature rose unaccountably to 102·2, and the next day to 103·2. She became, perhaps, somewhat more drowsy than she had been; and the ear that had been discharging presented at the meatus an unhealthy-looking sore. On the 25th it was noticed that she had lost power in her legs; and she moaned a great deal, as if in pain. In the evening she began to tremble violently, in arms, legs, head and neck, and trunk; but on the right side more than on the left. This continued for several hours. The temperature was then between 101 and 102. On the 29th it was observed that the legs remained weak, so that she could not stand; that she

choked a good deal when taking food; that the veins of the forehead and inner extremities of the eyelids (especially the latter) were greatly distended; that she seemed in pain, but was very quiet, and that her pulse was 92, very feeble and slightly irregular. She gradually sank from this time, and died at 5.15 on the morning of the 30th. Her temperature, which during the last ten days had varied a good deal, but on the whole had continued high, rose an hour before death to 105·4.

It is noticeable that the patient never complained of giddiness and was never sick; that her evacuations were always passed consciously; that she had no fits, unless the prolonged tremors which occurred a few days before death are to be regarded as a kind of fit; and that she had no pain in her head until shortly before her death.

Post-mortem Examination.—There was slight general congestion of the surface of the brain, with flattening of the convolutions and diminution of the subarachnoid fluid. The lateral ventricles were largely distended with fluid. The parts bounding these cavities were somewhat softened. Originating, apparently, in the valve of Vieussens was a soft, translucent, vascular new-growth, which extended into the neighbouring part of the cerebellum, mainly its middle lobe, and appeared to involve to some extent the corpora quadrigemina, which looked swollen and œdematous. It was adherent to the floor of the fourth ventricle, the ependyma of which was converted into a thick pulpy layer; but it did not appear to have encroached on the subjacent medulla. The growth altogether was perhaps about the size of a bantam-fowl's egg; but the limits between it and the nervous tissue, which it invaded, were very ill-defined. All other parts of the brain were healthy. Cord healthy.

Patches of dura mater, in both anterior fossæ, in both middle fossæ, and in a less degree in both posterior fossæ were the seat of growths identical with that affecting the brain. They formed irregular tracts, were a line or two thick, soft, translucent, slightly adherent to the surface of the brain above, and had caused erosion and honey-combing of the subjacent bone-tissue. Some of the nerves issuing from the skull seemed to have been

more or less pressed on by these growths; and it was believed that the affection of the ear observed during life was due to the affection of the dura mater.

The neoplasm appeared to consist of small oval and fusiform cells and blood-vessels.

Thoracic and abdominal viscera all healthy.

CASE III.—*Tubercular mass in third ventricle involving both thalami and followed by tubercular meningitis.—Tremors and rigidity of arms and legs; drowsiness; irritability; optic neuritis; slight facial palsy. Convulsions, coma, and elevation of temperature coming on with the meningitis.—Death.—Autopsy.*

Frederick P., aged 4, was admitted under Mr. Sydney Jones, for scrofulous disease of the left astragalus, which was excised on the 22nd of October, 1878. He went on very well until the 28th of January 1879, when tremulous movements were observed in the left arm and leg. These occurred only during the performance of voluntary movements, and consisted mainly in small to-and-fro movements at the larger joints, resembling those of disseminated sclerosis. There was slight loss of power in both of the affected limbs; but he could grasp with a fair amount of force. No other symptoms whatever, referable to the nervous system, were present. The tremors continued, but it was noticed that they were more severe on some days than on others, and that they ceased during sleep. Early in March tremors first showed themselves in the right hand; and slight spasmodic movements were observed on the left side of the face. All these symptoms continued, with little or no material change, and without the development of any fresh ones, up to the 12th of May, when he left the hospital.

He was re-admitted on the 8th of July. He had rapidly got worse, in respect of his ankle, his nervous symptoms and his general health; and latterly had become drowsy, and passed his evacuations unconsciously.

Face pale; a large distended vein runs transversely across the right upper eyelid. Very sleepy, and indeed seems always asleep, excepting when he is disturbed; and although his

appetite is good, and he has no sickness, he never asks for food; but he eats ravenously all that is given him. He constantly falls asleep while taking his meals, and has to be roused for each fresh mouthful. He does not seem to be in pain; but has general hyperæsthesia; is very peevish and irritable; and cries out when contradicted or disturbed. When nurses or others stand at his bedside, he attempts to ward them off with his right hand, and whines "No."

The mouth is natural when at rest, but is much drawn to the right when he laughs or cries, while the right eye becomes partially closed, the left remaining open. No squint; pupils equal, dilated, responding to light. Commencing optic neuritis; veins tortuous, no hæmorrhages. Tongue protruded straight, tremulous. Speech slow, but otherwise natural. No deafness. Head retracted somewhat as he lies. There are rhythmical tremors of the right hand and arm and leg, which are excited only by voluntary movements, and cease during sleep. They are much more pronounced than when he left the hospital. The movements on the left side have ceased; and the limbs have become rigid; the left elbow-, wrist-, and finger-joints are strongly flexed. The finger-nails are digging into the palm; and great pain is caused by the attempt to straighten the fingers. He often rubs or scratches the right side of the face. No dysphagia or vomiting; bowels constipated; motions passed unconsciously. Temperature ranging from 99·4 to 101. Pulse 114. Respirations 24.

During the next three months his condition varied little. He was drowsy; often seemed in pain; and was extremely irritable, crying out when pressed to answer a question. He could still see; but his sight was manifestly impaired, and he would apparently clutch at objects which did not exist. The tremors on the right side continued; and the left arm and leg remained rigid, and for the most part still. Slight tremors, however, were now and then observed in them. There was slight facial paralysis on the left side, but none of the tongue or ocular muscles. He had no vomiting, and no fits; and no evidence of disease in the chest or abdomen was obtained.

On the 8th of October he became worse, and vomited twice; and his temperature next day rose to 103·2.

On the fourteenth the following notes were taken. Has scarcely spoken a word since the 8th; and since the 10th his temperature has varied between 101·8 and 102·8. Yesterday afternoon he seemed fairly well, could see, and recognised his mother. But at 10 P.M. it was observed that his left pupil was much larger than his right, and that he took no notice of anything placed in front of his eyes. At 4 A.M. this morning he got unconscious; the left limbs became extremely rigid and tremulous; he foamed at the mouth; his eyes were directed upwards and to the left, and his eye-lids were blinking. He was still in this condition at 10 A.M. He had not called out, and had taken no food. He remained unconscious during the rest of the day, lying with head strongly retracted, breathing stertorously, and having every few minutes attacks of rigidity, affecting both arms and legs, but limbs of right side mainly. Drops of sweat collected on the face, and the right arm and leg became notably cold; but the temperature of the body rose to 103·2. He was unable to swallow.

Death took place at 4 A.M. on the 15th.

The eyes were examined shortly before death, when the veins at the back of the left were found dilated and tortuous. The right presented recent ulceration of the cornea.

Post-mortem Examination.—Much emaciated; pupils of equal size. Old cicatrix over inner side of left ankle.

Meningeal vessels congested; pia-mater on vertex natural; convolutions flattened. Interpeduncular space, pons, and medulla, opaque and covered with flakes of recent lymph. A few miliary tubercles in membrane at base; and an abundance of them in the right fissure of Sylvius. Much distension of lateral ventricles with clear fluid. The third ventricle was occupied by an irregularly globular tubercular mass, about 1½ inches in diameter, which partly involved both optic thalami. Another small tubercular mass was found on the internal surface of the left occipital tube.

No tubercular or other disease was discovered either in the thoracic or in the abdominal viscera.

CASE IV.—*Tubercular mass in left lobe of cerebellum.—Effusion of fluid into ventricles.—Inability to walk; tremors of arms and head; optic neuritis and blindness; frequent epileptiform attacks; headache, &c.—Death.—Autopsy.*

A. G. T., a boy four years of age, came under my care on the 30th of December, 1879. About three months before that time he was suffering from hooping-cough and measles, and then had several fits. About two months before admission the mother noticed that he squinted; the fits also became more frequent, and he began to complain of pain at the back of the head.

He is a plump, healthy-looking child; seems quite sensible; answers questions, but speaks indistinctly, and drawls out his words very slowly. He can move his legs, but is unable to walk, or even to stand. His hands and arms (especially the left) tremble when they are in use, and there is apparently want of co-ordinating power in them. His head and trunk also tremble when he sits up. He has no squint, and the pupils are dilated and equal. He appears to be completely blind. There is no definite paralysis, no impairment of sensation, and all the reflexes are well-marked. The pain in his head comes on in paroxysms, and is referred to the occipital region; and, when he is lifted up, he screams out, and puts his hand to the back of his head. There are no signs of disease in the thoracic or abdominal organs. Tongue clean; appetite good; bowels confined; urine 1020, urates and phosphates, but no albumen.

His symptoms underwent very little change during the next few months. He remained sensible, but spoke slowly and with difficulty; he suffered at times from severe headache, which often made him cry out, but in the intervals he was remarkably good-tempered, and always ready to laugh; the weakness of the legs, and the tremors of his head and neck and arms continued; and he passed his evacuations into the bed; he retained a fair appetite, and had no sickness. He had frequent fits, sometimes several in the day; the period of insensibility varying from two minutes to a quarter of an hour. In some of them he was quite still, save for a little twitching

of the eyes and eyelids, and turning in of the thumbs. In the more severe ones, the arms and legs became rigid, the former being raised above the head, the latter being straightened, with toes pointed; but the rigidity was more marked in the right than in the left arm, and the latter generally became limp during the progress of the attack. The fits were occasionally ushered in with a paroxysm of crying. On the 23rd of January his eyes were examined by Mr. Nettleship, who reported that he thought the boy had perception of light with right eye, for he followed the mirror with it, and the pupil acted fairly to light; that the left pupil also acted, but irregularly; and that the last stage of optic neuritis (woolly discs), passing into atrophy, was present in both eyes, without hæmorrhage, or much swelling. His head was large, and it was suspected that it was increasing in size; its measurements were taken therefore early in March and again early in April; but no progressive change was discovered.

At the end of March, or the beginning of April, he began to suffer from drowsiness; and was becoming manifestly more and more feeble-minded; much less inclined to speak, and also much less inclined to laugh. Further, he often had, besides his stronger fits, apparently momentary attacks of loss of consciousness, and the limbs were often rigid. The surface was apt to become flushed. The tremors of the head and neck and arms continued. His appetite became impaired; and he had difficulty in swallowing, more especially solids.

During May he became very quiet, ceased to speak, often refused food, and it was observed that he did not close the left eye so perfectly as the right, and that it presented a slight internal squint. There was no doubt that he was now quite blind; and the pupils, which were equal and dilated, did not respond to light. His face was now without expression. The circulation was very imperfect, and the hands were cold and livid. He had become very thin.

During the next two months he passed mainly a vegetative existence. He lay almost unconscious, occasionally moaning, but never speaking, though even to the last apt to present a flickering smile when tickled. His arms became more or less permanently stiff, and in great measure ceased to tremble.

Late in June it was observed that the left conjunctiva was insensible, and that an ulcer had appeared in the cornea. This proceeded to perforation and prolapse of iris, which took place about the 14th of July; at which time an ulcer had appeared in the right cornea also. It may be added, that early in June some vesicles made their appearance on the cheeks and forehead, which subsequently became pustular, and that one of them reached the size of a shilling. He vomited once or twice during the month of July. His death had been anticipated daily, for weeks before it actually occurred on the 6th of August, and was due apparently to a combination of coma and asthenia.

Post-mortem Examination.—Much emaciated; head very large, anterior fontanelle slightly open.

Surface of brain dry; convolutions flattened. The ventricles were greatly distended, containing about two pints of watery fluid. The brain generally was soft, but otherwise healthy. A small tubercular mass was imbedded in the grey matter of the orbital surface of the right frontal lobe; and another mass of similar nature, and about the size of a Tangerine orange, occupied nearly the whole of the left lobe of the cerebellum. The brain-tissue around was softened, and the part in contact with the tentorium was adherent to it. The part of the left cerebral hemisphere that was in contact with the diseased cerebellum was superficially softened. And the corresponding side of the pons varolii and medulla oblongata were pressed upon and somewhat softened. There was no evidence of implication of the ganglia at the base of the brain. The optic nerves, especially the left, were small. Heart and pericardium healthy. Lungs collapsed below, but otherwise pale and healthy. Glands at root caseous. Chylopoietic viscera healthy. Some pyelitis of both kidneys, with a few small dark-yellow concretions.

CASE V.—*Chronic Hydrocephalus.—Dropsical accumulations in lateral and third ventricles, in fourth ventricle, and in central spinal canal.—Weakness of intellect: stiffness of neck: feeble, tottering gait: epileptiform fits.—Death—Autopsy.*

B. S., a harness-maker, aged 17, was admitted under my care on the 8th of June, 1872.

He was said to have been a healthy lad until about four months ago, when he began to complain of pain in his back when at work, of headache and nausea. Three months ago, while at church, he was seized suddenly with pain in his back and head, and giddiness so severe, that unless he had been supported he would have fallen. From this time the pain in his head was so intense and constant, that he was compelled to give up his employment. He remained in this condition up to three weeks ago, when he had three epileptic fits. On the 3rd of June he had two others, but did not lose consciousness in them; and for a short time subsequently he had no use of his right side. Since his illness he has suffered from constipation; and has occasionally seen double.

He is is a well-grown, but dull, heavy-looking, large-headed youth; apparently slow of intellect and speech; and slow and unsteady in his movements, yet with no definite paralysis. He has no discoverable disease of the thoracic or abdominal organs. Pulse 84. Temperature 98·6. Urine 1008, free from albumen.

While he was under observation in the hospital, the following circumstances were noted. The patient has no paralysis of any of his voluntary muscles, and no affection of his tactile or other senses. And he has complete control over his rectum and bladder. But his neck seems stiff, and he has manifest difficulty in rotating his head from side to side, and upwards and downwards; he walks feebly and unsteadily, and with a shuffling gait. His mental condition is peculiar; and gives the impression of slight congenital imbecility, which, however, is denied by the friends. While in the hospital, he was occasionally noisy at night time.

On the 22nd he had a fit. On the 28th he complained more

than he had previously done of headache. During the night of the 29th he was more noisy than he had been; but otherwise seemed much in the same state as usual. Early in the morning of the 30th, he became suddenly livid in the face, and insensible, and died without any convulsions in the course of a few minutes.

Autopsy.—Head 23 inches in circumference externally. Skull very thin, especially near sutures, and distinctly marked by convolutions. Dura mater healthy. Pia mater injected. Surface of brain dry; convolutions flattened, and fissures almost obliterated. The infundibulum was much distended with fluid; and on removing the brain, clear colourless fluid poured away from this part. The basal portion of the brain, from the crura cerebri to (and including) the lamina cinerea, was distended with fluid, and not thicker than paper. The fourth ventricle was also clearly distended with fluid, and its lateral margins formed on either side a saccular projection just behind the crus cerebri.

On laying open the lateral ventricles, they were found to be largely and uniformly dilated. Many ounces of fluid had escaped; but several still remained. The communication between them, and between them and the third ventricle, was very free. No septum-lucidum, properly speaking, could be found; and the fifth ventricle appeared to be represented by a triangular prolongation of the third ventricle forwards. The corpora-striata and optic thalami of the two sides were widely separated by the large and distended third ventricle. The commencement of the iter was quite blocked up by a translucent septum, looking like a portion of the ependyma. The testes and nates and roof of the iter were together much widened, bulged upwards and very thin, evidently from distension of the iter with fluid. The valve of Vieussens, and processus e cerebello ad testes, were also greatly distended and tense, forming a fluctuating tumour which was continuous with the projections observed behind the crura cerebri. The pons itself looked normal. It was clear that the fourth ventricle and iter formed a common cyst, isolated from all other cavities. The substance of the cerebrum, cerebellum, and other parts of the brain, was of normal consistence and struc-

ture. The lining-membrane of the ventricles was somewhat thickened.

The medulla oblongata and the first six inches of the spinal canal were much enlarged, leaving scarcely any space for subarachnoid or arachnoid fluid. This was due to dilatation of the central canal, which was full of serous fluid. There was no communication above between this and the fourth ventricle; and below, the central canal became, as in the normal condition, inconspicuous.

There was no inflammation, tubercle, or new growth of any kind. The nerves at the base of the brain appeared all healthy.

The weight of the brain, including the fluid (perhaps an ounce) in the fourth ventricle, was 66½ oz.

All the other viscera were healthy.

A STUDY OF A CASE OF EPILEPSY.

BY CHARLES MERCIER, M.B.

ELIZABETH C., æt. 29. Family history presents no data of importance. Her first fit occurred at the age of nine, and she has had fits at irregular intervals ever since. Her own account is that she knows when the fit is coming on, that is to say, "she can feel it about her," but she cannot tell when it "takes her." She cannot describe the pre-paroxysmal feeling. "It is too horrible." "It is in her chest, and head, and all over her, but in her chest and head most. It lasts an hour, or sometimes two hours." Before witnessing her paroxysms I placed no reliance on this statement, believing that she had much overstated the duration of the aura; but, as will be seen, the actual duration was very much longer than she describes.

During the nine months that she has been under observation, her fits have occurred in batches of from 4 or 5 to 30 or more, the batches being separated by intervals varying from a few hours to as much as three months. The fits and batches of fits spread over this period varied somewhat in minor details, but in their general characters maintained a remarkable uniformity.

At the outset of every batch of fits the first noticeable occurrence usually is, that the patient becomes captious and irritable, quarrelling with all her neighbours without reasonable provocation. When questioned at this time she answers defiantly and insultingly, in a manner quite alien from her usual demeanour. At the same time, or soon after, she begins to complain of pain, often definitely localised in the side or shoulder-blade, or epigastrium, and later of "horrible feel-

ings" in her head and chest, and all over her. The look of distress that her face has borne hitherto now deepens into an expression of mingled sulky ferocity and anguish; and as time goes on, the latter expression predominates and the former fades out. Next she becomes taciturn, answers impatiently and indistinctly; her articulation is first slovenly, and then decidedly defective. She appears overwhelmed with unutterable languor, her limbs are flaccid and her attitude one of limp helplessness. Her answers come at longer and longer intervals, and at length are elicited only after repeated and urgent questioning; and she no longer looks at you when she speaks. About this time it is noticed that her face has assumed an earthy tint; this is a very marked feature. The complexion is something like that of advanced Bright's disease, but is a deeper and more muddy hue. Her expression becomes more agonised, but has an added and increasing look of vacancy, and at length a definite strabismus appears, marking the period when answers can no longer be obtained, although a trifling movement of the lips or a faint gesture of despair shows that consciousness is not altogether lost. When this stage is reached, the onset of convulsion is not far distant; indeed, it may be said to have already begun, for upon examination at this stage the hands and forearms will be found to be already somewhat rigid, and to have assumed a position approaching that which is characteristic of the paroxysm.

The foregoing series of events is spread over a time that varies on different occasions, but is always considerable. On Oct. 9, 1882, a note was made:—The shadow of the coming fit is apparent for a long time before the fit occurs. Yesterday her eyes had lost their brightness and looked lack-lustre, and a dark areola was noticed round them, five hours before the fit began; and at least three hours before the fit her articulation became worse, she was very languid, and had a feeling which she describes as "horrible." Two hours before the fit, she lay prostrate, and could scarcely be roused to answer, and when she did so, it was found that her articulation, previously fairly distinct, had become almost unintelligible. She could just say that she felt "fitty." On Oct. 29th, at 10.30 A.M., it was noticed that "Her face has an earthy tint, her eyes a sunken

appearance, with a dark areola round them, such as is usually noticed before the onset of her fits...she says that she feels 'very indifferent,' and 'as if the fits were coming on,' &c. About 10 P.M. the same night she began to be convulsed. *Dec.* 2.—"After a long intermission of fits, none having occurred since Oct. 29, she began the day before yesterday to complain bitterly of feeling 'fitty,' and of a dreadful feeling in her head. Her articulation at the same time became more imperfect, her eyes sunken, her face earthy, her expression distressed, and after a time a slight squint appeared. She became gradually worse for fully twelve hours, and then began to have fits." The most remarkable instance of prolonged warning is, however, the following. On Feb. 6, 1883, I made a note: "She is less animated than usual this morning, her face has a somewhat dull expression..., &c. It is probable that a fresh batch of fits is coming on." Confirmatory notes were made on the 7th and 8th, and on the afternoon of the 9th a further note: "Her face has a vacant expression, and she squints slightly. When spoken to, she makes an effort to answer, but her words run into one another, and her articulation is so imperfect that it is very difficult to make out what she says," and so forth. It was not until the night following this day that the fits at last occurred; a distinct warning having been observable three clear days and a half before their onset.

At length a definite convulsion occurs, and this event is repeated at intervals of from a few minutes to a few hours, from four or five to twenty or thirty times, when the batch comes to an end.

The several convulsions of a batch are superposed on a condition of rigidity, which begins gradually some time before the first fit, towards the end of the stage of warning; continues without intermission in the intervals between the several fits of a batch; and slowly disappears some time after the last fit is over, leaving a condition in which losses and defects are conspicuous. On one occasion the hands were distinctly rigid at 5 P.M., the last fit having occurred at noon. The position imposed by this rigidity is one of extension and pronation. The head is thrown back, the body is over-extended or

opisthotonic, the legs and feet are extended, the arms are by the side of or in front of the trunk, the elbows extended wholly or three quarters, the forearms very strongly pronated, and the hands clenched. The eyelids are moderately open and the eyes to right. The position is well shown in Plates I. and II., save that in the former the extension of the head was artificially diminished, with the intention (unsuccessful) of showing the position of eyes and mouth. The rigidity is greatest in the hands, in which it cannot be overcome by any justifiable amount of force; less in the wrists, still less in the elbows, and not very great in the shoulders. It is less in the legs than in the arms, or neck; the head being retracted with a force approximating to that which immobilises the elbows.

Besides the rigidity, there is another motor phenomenon which is never absent in the inter-paroxysmal stage. At the beginning of every inspiration the left corner of the mouth is suddenly drawn downwards and outwards to an extraordinary extent, the commissure of the lips being at least doubled in length with each such spasm. The rest of the mouth remains unaffected, save in so far as it is disturbed by the dragging of this corner. This movement is mainly due to contraction of the platysma, which is seen to be in violent action, and stands out prominently with each spasm. But the whole jaw moves slightly to the left, and the masseters unquestionably harden at the same time. The spasm of the mouth is invariably synchronous with the respiratory movements. It begins at the same moment as inspiration, continues as long or nearly as long as inspiration lasts, and as the inspiratory movement reaches its height the mouth returns to its symmetrical shape. When, as often happens, inspiration is uniform—when its evenness is interrupted by slight checks, such as may be noticed in the long-drawn sighs of a sobbing child, a corresponding wavering occurs in the spasm of the mouth. With expiration, the lips are puffed outward and vibrate with a purring noise. At the end of expiration there is an interval of quiescence, which is often considerably prolonged, before the inspiratory movement begins; and during this interval the mouth is quiescent. Then the cycle begins again. These movements I have witnessed several scores of times, and have

Woodburytype.

PLATE I.

never seen any variation from the order and combination described. I have notes of another case of convulsion in which an analogous association of spasm with the respiratory movements occurred. In that case also the spasm so associated occurred in the ebb intervening between two tides of convulsion. The spasm took the form of a couple of twitches, —two shocks of "simple contraction"—separated by a brief interval and affecting simultaneously the face, head, arms, and legs. This pair of contractions occurred invariably at the end of every expiratory act,—at the beginning of the pause which precedes inspiration in the slow breathing of coma, and was observed more than one hundred times.

Returning to the case of Elizabeth C.; on one occasion a third motor abnormity was noticed in the inter-paroxysmal period of the convulsions. After a free interval of two months she had had a batch of fits extending over twenty-four hours; then an interval of twelve hours, and then a single fit. After this fit, she was lying on her back, the arms, legs, and neck being rigid as usual. Breathing was slow, deep and laboured, the mouth drawn in inspiration, as above described, and the lips puffing in a flaccid manner that showed deep coma. A rather unusual amount of movement of the chest in respiration led to further examination, and it was then found that the diaphragm was not acting. There was paralysis, total or almost total, of the diaphragm. While my hands were placed on abdomen and chest, for the purpose of examining this matter, I felt a subdued quivering beneath them, and this gradually increased, until at length she was shaking all over with a fine tremor of about 240 to the minute, like a person in a rigor, or shuddering with cold. There was no marked cutis anserina, and the condition of the pupils was not observed. The movement appeared to begin and be most pronounced in the thighs and about the pelvis; but it affected the whole body, as far as could be observed; and when the left forearm, which was not visibly tremulous, was grasped, the muscles therein were found to be quivering in unison with those of the legs. Passive movement increased the tremor, which continued for fully ten minutes, until a fit came on, but did not increase and merge into the fit. In connection with this occurrence it

should be mentioned that on one occasion, after a paroxysm, there was tremor of the right arm "as if from fatigue."

The paroxysms of convulsion that are superposed on, or grow out of, this rigidity, are much alike in general character, although they vary a good deal in minor details. In all the spasm is of tonic quality. The movements are, as a rule, slow, flowing and continuous; or if there is a twitching, it has never the electric suddenness of clonic spasm, and the separate simple contractions are never perceptible. The onset is always deliberate. The spasm always begins in the periphery and spreads towards the trunk. It always begins bilaterally, and remains so; but its invasion is rarely quite simultaneous on the two sides, and there is always a preponderance of the spasm on one side,—the right being the first and most effected. Shortly after the onset of the fit, she rolls over to the right and lies on her face, in the attitude given in Plate III. At this time the thorax is locked in rigid spasm, the face is extremely livid, but is not much distorted, and in the otherwise rigid body various large and general movements occur. Subsidence is never by clonus; but the cessation of the spasm of the glottis is announced by a groaning cry, and as the pent-up air is at last expired, a general relaxation occurs, and the body subsides into an attitude of comparative repose; but, on examination, this relaxation is found to have been only partial, the rigidity already described remaining.

The origin of the convulsion, though always peripheral, never, that is, beginning in the bilaterally acting muscles, is yet variable; or rather it would be more correct to say that it begins almost simultaneously in eyes, mouth, and hands, but that the simultaneity is not exact, and that which of these parts shall have a momentary precedence is a quasi-accidental matter. In one paroxysm the first thing noticed is an increasing rapidity of those twitches of the left corner of the mouth that have been described, respiration at the same time becoming imperceptible. Other fits begin by turning of the eyes; but in this case again the ill-defined localisation of the onset is seen in the fact that the first movement of the eyes is often to the left, and not until after a few moments of wandering do they take up their position to the right. The movement of

PLATE II.

Woodburytype.

PLATE III.

the hands at the outset of the fit is not, as is usual, a gradual closing up into a knot of spasm, for the hand is already rigid, but a to-and-fro movement of pro- and supination (pronation being the active element), which is at first about five or six to the minute, and not very sudden, and becomes more and more rapid, until it ends as continuous and extreme pronation.

While there is a general bilateralness of the convulsion, yet the preponderance of the spasm is always on the right. However the eyes may be at first directed, they soon turn to the right, and remain so. The right hand is often affected before, always more than, the left. In every fit she invariably, however she may be lying, turns over and lies on her face, the rotation being always from left to right. Once this attitude is attained, no further one-sidedness of the movements is apparent; but that the right suffers most is shown by the enduring paralysis of this arm and leg, the left recovering rapidly.[1]

At the height of the fit, when the prone position is complete, large general movements of the whole body occur. These often take the form of violent and extensive to-and-fro movements of the pelvis. On several occasions there were very definite alternate movements of the arms and legs,—movements as of quadrupedal locomotion, which, taken in association with the prone position, were very remarkable. They vividly reminded me of the movements of a choreic dog.

Plate III. is an instantaneous photograph taken on one of these occasions. On other occasions the movements were tumultuous tossings about, of indefinite and indescribable character. In every case, however, the electric suddenness of clonic spasm is conspicuously absent. The force of the movements is amazing.

The recovery from the state of exhaustion and coma in which she is left by the last fit of a batch is at least as gradual and prolonged as the onset of the attack; two, three, or more days often elapsing before consciousness can be said to be fully regained; and even when she has recovered in this respect,

[1] While this is in the press, she has had for the first time a batch of fits in which she turned to the left, and the left arm remained paralysed and rigid after recovery.

various sequelæ are found to remain behind. When she first came under notice, in June 1882, the right leg was weaker than the left, and this weakness increased after each batch of fits. After a prolonged attack on Oct. 1 and 5, the right leg remained completely paralysed and anæsthetic. On Oct. 11, after another attack, the right arm remained paralysed, anæsthetic, and rigid in the position of the late rigidity of hemiplegia. This condition remained nearly stationary for six months, and has only lately improved. The most remarkable of the post-epileptic losses, was, however, the following.

After a succession of batches of unusually numerous and severe fits at the end of August 1882, her recovery was very slow. Even on the third and fourth days she appeared dull, heavy, and stupid, and responded to questions only by faint and impatient gestures. As she gradually regained her intelligent expression and her consciousness, she still continued to express herself by signs alone, and it became manifest that she had lost verbal utterance. Her inability to utter a single word, coupled with her evident distress, and the manifest efforts that she made to speak, was at first supposed to be aphasia; but it very soon appeared that she was quite able to write, and to express herself fluently and correctly in writing. The loss was of articulation merely. In the intervals of her convulsions she improved; but each fresh attack threw her back, and reduced her to a more or less totally inarticulate condition, the defect extending altogether over about three months, at the end of which time a long freedom from convulsions allowed of her complete recovery. After fits subsequent to this recovery, the return of articulation has been delayed beyond the return of consciousness, but no prolonged articulatory defect has existed since.

The following note describes her condition at the lowest ebb of this special loss; at that period after the paroxysm when consciousness appeared to have completely returned, but the defect of articulation was still extreme. *Sept.* 15.—"She sits up in bed, looking about her intelligently, and showing much interest in what is going on. She smiles, showing no paralysis of face; she cannot whistle, never having acquired this accomplishment, but she readily pouts her lips into the

appropriate position. She kisses (her own hand) quite perfectly. She puts out her tongue when told, and performs adroitly every movement that is indicated to her. She makes, without the least perceptible defect, the clicking noise familiar to coachmen, and utters rapidly the 'tut tut' of impatience. She can also clear her throat with a sonorous hawk. But although she speaks fluently, she cannot articulate a single word. Her utterance consists of only one vowel, without any consonants at all. Imagine every vowel sound in every word to be replaced by the single sound *ah*, or *ăh*. Imagine every consonant to be discarded, and replaced by a mere momentary break in the utterance—a break formed, not by any movement of lips, tongue, or fauces, but apparently by a closure, or partial closure, of the glottis, and a respiratory check. Imagine the words thus degraded to be uttered rapidly, vivaciously, with a very varied cadence and abundance of vocal expression; and you have the utterance presented. While the lips, tongue, jaw, palate, and fauces, appear to be absolutely passive, and to take no part in articulation, her words, if words they can be called, can often be recognised, and her meaning apprehended, from the rhythm of the utterance, taken in association with the cadence, and regard being had to the probable nature of the propositions that she attempts to express." The various consonants and vowel sounds were carefully gone over, the general result being that labials were sounded worst, dentals more successfully, and gutturals, after several efforts, fairly well. All the vowel-sounds were first uttered as *ah*, but after a good deal of practice, she could, after beginning the required sound as *ah*, subsequently modify it into the sound required. Thus *ee* was uttered as *ah-ee*, *o* as *ah-o*, &c. At this time she could write quite fluently, but with curious omissions of words, a defect that was not aphasic, but peculiar. For instance, I had been speaking of sending her back, and she wrote: "I don't want you—me back," the words "to send" being omitted. Subsequently she wrote, "You don't feel—but I do. I never be like this. Any one would have—told me this six months ago." Her meaning doubtless was, "You don't [know what I] feel, but I do. I never [would have believed that I should] be like this, [if]

any one would have told me this six months ago." After some days, however, this defect disappeared, and she wrote her history at length, quite intelligibly, while she was totally inarticulate. While writing, she made grimaces (she is unused to writing), but did not form the words with her lips.

It is unnecessary to record the fluctuations that from time to time occurred in her power of articulating, but it may be said generally that the vowels always returned before the consonants, that the sound *ah*, was always the most persistent form of utterance, and that the consonants showed no regularity in the order in which they were regained. Labials returned sometimes after the other consonants, sometimes first of all. Occasionally she could utter a sound, especially a dental sound, at the beginning of a word, when she could not utter it as a terminal. On one occasion she totally lost even the rudimentary sound that usually represented her articulation, and while she indicated her appreciation of what was said to her by doing as she was told, she failed to produce even the remnant of a reply.

On the morning of the 9th of October a note was made that she had almost entirely regained her articulation, and details in proof of this statement were taken. In the afternoon, however, she displayed a defect of more complex character which was not present in the morning. She was somewhat aphasic. "The words that she does utter are better said than usual, but before they issue there is a long pause, and an appearance of great effort; and there is a violent expiration in getting them out. Some words fail to be uttered even in caricature, and efforts to utter them end with the shake of the head and the despairing gesture common in aphasia. She has always been very voluble in her own jargon, but now is silent. On being asked a question, she says 'I—I—' and then a despairing gesture. Some words are uttered after many repetitions of the initial letter and with exaggerated emphasis, as in stammering. She appears to think for the right word, and to be unable to find it, like an aphasic. She understands clearly, however, a rather intricate pun." On the evening of this day she had several convulsions.

Shortly after this the right hand remained, after some severe

convulsions, rigid, and therefore useless, and on being asked to write with her left hand, she wrote fluently in mirror-writing. It appeared, however, that she had learnt this before. Although not herself left-handed, her maternal grandmother was so, and her own mother was ambidextrous.

It was noticeable that at a time when her articulation was quite normal at the beginning of a conversation, it would fail towards the end, when she got fatigued.

Comments.

In this remarkable and instructive case, of which the main features only are enumerated in the foregoing account, the points that most call for consideration are three; the prolonged warning, the character of the convulsions, and the articulatory defect.

Taking first the articulatory defect, and first of all the intrinsic relations of this defect, stress must be laid upon the fact that, speaking generally, and allowing for aberrations in detail, the occurrences followed the law laid down by Dr. Hughlings Jackson—the special arrangements suffered most and returned latest, and the general arrangements suffered least and recovered earliest. That the vowel-sounds have a wider generality than the consonants is sufficiently indicated by the fact that the former are capable of standing alone as integral articulatory wholes, while the latter are merely accessory and have scarcely an existence, save as limiting a vowel-sound. The first articulatory element to appear in the infant is simple phonation—an undifferentiated vowel-sound. The first element to become firmly established in civilised races is the broad *a*, whose fundamental quality is shown by its forming the staple of the first articulate utterances, "da-da" "pa-pa," &c. Not until the arrangements for this sound have become to some extent organised, do the other vowel-sounds appear.[1] That consonants appear later still is seen not only

[1] That the early appearance of this sound is not peculiar to our race, and not, therefore, a quality that has been lately acquired, is shown by the facts of comparative philology. For I find that out of 417 languages, the predominating vowel-sound in the word for "father" is *â* or *a* in no fewer than 315, or more than three-fourths; while out of 316 languages the predominating vowel-sound in the

in the developing child, in whom some of them often remain undeveloped for several years, but in the fact that the lower animals can scarcely be said to possess any consonants. Now in the case here recorded, the vowels had completely returned at a time when the consonants were still defective; the former were partly retained at a time when the latter was wholly lost; and of all the elements of articulation the broad *á* was the first to be recovered; the other vowel sounds being subsequently acquired by gradual modifications of it. As to the consonants, there was no constant order of recovery, but neither is there any constant order of acquirement, nor could any consonant be said to be more highly special than the rest. Occasionally she was able to articulate a word perfectly in its appropriate place in a sentence, although she failed to utter it when told to do so. The corresponding defect, which is common in aphasia, has been pointed out by Dr. Jackson to be the loss of the most voluntary and special mode of using the word.

As to the extrinsic relations of the loss of articulations—its relations with the condition of the neighbouring faculties—the most notable circumstance was the isolation of the defect. Not merely was there no paralysis of the muscles involved, not merely were all the other buccal, lingual, &c., movements normal, but at a time when speech, as indicated by writing, was perfect, and cadence was not in the least impaired, articulation was altogether lost. Are we then to conclude that the faculty of articulation is localised in a region of the grey matter distinct from those appropriated to speech and cadence? The dictum against multiplying causes should render us very re-

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word for "mother" is the same in 190, or nearly two-thirds. As it is often difficult to infer the correct sound from written words, I left out of this computation all cases in which the sound appeared doubtful. Taking the facts in another way, the fundamental character of the sound becomes still more apparent, for out of 417 languages there are but 42, or one-tenth, in which the vowel *a* is not used in the formation in English writing of the word for "father;" and out of 395 languages there are only 72, or between one-fifth and one-sixth, in which the same vowel is not used in the formation of the word for "mother." Had languages of Aryan stock alone been considered, the proportion would no doubt have been greater. The facts were extracted from Professor Buschmann's tables.

luctant to think so. Yet many facts indicate a certain degree of solution of the common matrix from which they have unquestionably been differentiated, and that they cannot be looked on severally as mere grades in the larger faculty of language appears certain. Were they so, not only should all be disordered concomitantly, but, according to the law above quoted, speech should always be affected first and most, articulation less, and cadence least. But in paralysis agitans the very reverse obtains. Cadence is lost, articulation is slightly affected and speech is normal. Moreover, the facts that a person can read fluently the words of a language that he does not understand, and that, as in the case related by Coleridge, a person in delirium can repeat pages of a foreign tongue of which she does not understand one word, appear to demonstrate beyond question that articulation is largely separable from speech, and that the two may therefore be disordered independently. In the present case, although articulation suffered by far the more severely, yet there is evidence that in the onset speech was affected first. Aphasia is the loss of the physiological activity of a certain tract of the highest nervous arrangements, and when this tract alone is thrown out of action, the defect shows out conspicuously by contrast with the unimpaired activity of the surrounding nervous arrangements. If we look on the physiological basis of consciousness as a continuum of activity throughout all the highest nervous arrangements, then if this activity be progressively diminished in all these centres simultaneously, there will be a progressive degradation of consciousness in all its forms; and the defect of any individual faculty will be merged in the general loss, and may be overlooked. In this way the aphasia that was unquestionably present in the prolonged stage of invasion was rendered inconspicuous, the loss of a part being drowned in the loss of the whole. On one occasion, however, in the gradual degradation of processes that occurred before the fits, the defect in the language series was sufficiently in advance of that in the others to be separately recognisable, and accordingly a condition of unmistakable aphasia resulted, as detailed in the note of the 19th of October. Granting, however, that speech was first affected, it must nevertheless be admitted that

it was not affected most severely, and it certainly recovered long before articulation; so that while there is evidence that the discharge began according to rule in the highest centres, yet it must during the convulsive stage have assumed an overwhelming preponderance in centres comparatively low in grade, and that this was so is indicated by all the localising characters of the convulsions themselves.

To recapitulate these characters:—The spasm was of tonic quality. By tonic spasm I mean spasm in which the constituent "simple contractions" never become discrete and separately perceptible, but are fused together, as in this case, into waves of gradual rise, progress, and decline. Next, the spasm was bilateral and approximately symmetrical. Beginning deliberately, it was seen to invade both arms independently and almost simultaneously, and to travel centripetally from the hands up to the trunk; the attitude produced was one of universal extension, and in the forearms of pronation also, and this attitude was the same in both sides. If we remember the characters of Jacksonian epilepsy—convulsions which are clonic from the outset, which always begin unilaterally, and commonly in one thumb and finger, which often remain throughout conspicuously unilateral, which never produce a symmetrical attitude on the two sides, in which flexion invariably and everywhere preponderates, and the forearms are commonly supinated, we shall see that they are in almost every respect the reverse of the convulsions in this case, the only common feature being the centripetal march. And the convulsions of Jacksonian epilepsy are, we know, due to discharging lesions of the cortex of the cerebrum. Hence the inference is valid that wherever the discharging lesion is in this case, it is not in the cerebral cortex. Again: the excessive discharge of a region of highly elaborated centres is necessarily attended by the abolition of the function of the parts represented. When a cerebral region is so discharging, the several movements that should normally occur separately in orderly succession are all jammed into the same moment—all strive to occur at once—and the result is a chaos of spasm. But when a region represents but a single undifferentiated movement, the discharge of that region, however excessive,

however rapid, can cause only that one movement, which is still therefore a co-ordinated movement, though it is excessive in degree. For instance, when the vaso-motor region so discharges, the normal movement—contraction of the arteries—still takes place, as we see in a rigor, although of course it is excessive in amount. Applying this consideration to the case of centres intermediate in grade between the most elaborate and the simplest, it appears that we can judge approximately of the grade of a centre that is discharging excessively, by the amount of co-ordination that exists in the resulting movement. Now in the present case the outcome of the discharge was not a mere chaos of rigid spasm. There was a distinct co-ordination of a simple kind remaining, as was evidenced in the movements described as resembling those of quadrupedal locomotion, in which all four limbs were concerned, and moved in times definitely relating to each other.

(I have seen two other cases in which definitely co-ordinated movements occurred during the actual progress of a convulsion. In both cases the convulsions affected first and most the bilaterally-acting muscles, and the attitude produced was one of general extension. In one case violent and repeated sneezing (note the connection of the spasm with the respiratory function) occurred both at the outset and at the close of the fit. In the other the patient, while at the height of the fit, used to bite the back of her hand. The first of these movements is so deeply organised as to have become reflex; and the second was no doubt a caricature of the action of carrying food to the mouth—also a movement, which, though much more elaborate than that of sneezing, is yet very deeply organised in the race.)

The tonic quality of the spasm, the continuous duration over many hours, the symmetrical distribution, the attitude of extension, and the rotation which was so prominent a feature in every fit, and which never failed to occur, all powerfully suggest that the spasm was of cerebellar origin; but, on the other hand, the centripetal march of the spasm seems to me to outweigh all this evidence, and other circumstances reinforce this objection and appear definitely to negative such a supposi-

tion. The occurrence of anæsthesia is not perhaps of much weight, since it may have been due to exhaustion of some lower tract to which the spasm had extended. More consideration is due to the fact that the position of the arms in post-hemiplegic contracture and in tetanus, both of which are believed by Dr. Hughlings Jackson to be due to cerebellar influx, is one of flexion and supination, while here the arms were extended and pronated. But the most fatal objection is the existence at the height of the convulsion of those definitely co-ordinated movements already referred to as having a distinct, if a caricatured, resemblance to those of quadrupedal progression. The tendency of recent thought as to the functions of cerebrum and cerebellum is more and more towards the view that neither of these organs alone can affect a co-ordinated movement. The cerebrum is powerless to bring about such a movement in the absence of the underlying tone pre-existing in the muscles by virtue of their cerebellar supply. The cerebellar discharge alone can produce merely a static and enduring contraction which must be broken up by the interrupted discharge from the cerebrum before a co-ordinated movement can occur.

On the whole, then, it appears very unlikely that the paroxysms of convulsion in this case can have been directly due to discharge either of cerebral or of cerebellar regions.

If then the discharge was neither cerebral nor cerebellar, what was its origin? The simultaneous invasion of both hands implies that the discharging region was below the great bifurcation of the pyramidal tract, and the balance of the evidence favours the view that the main focus was at the upper part of the pons varolii. The very low grade of this region, compared with that of the cerebral and cerebellar cortex, and the fact that fibres from both these latter regions are here in close proximity, will account for the slight co-ordination that existed in the excessive discharge. The nearness of the middle peduncle of the cerebellum and its extension in the transverse fibres of the pons will account for the rotation that occurred. The proximity of the respiratory tract will account for the definite connection with respiration, and the existence

of anæsthesia and of the articulatory defect are easily intelligible if we admit the main focus of the discharge to have been in this region.

Seeing that the hypothesis of a pontic origin will account for the main features of the attack, let us ask if there are any features with which it is inconsistent. There is one which appears to offer a fatal objection. I have endeavoured to show in previous numbers of "BRAIN" that a discharge never does and never can begin, save in the highest centre of a series; and the centres here discharging were admittedly of very low grade. Discharge of such a region from general causes is only possible on the assumption that all the superposed centres were non-existent, as far as function is concerned—that they had all been previously discharged. Yet there were no convulsions antecedent to those of the lower region, to indicate previous exhaustive discharge of the higher. Nevertheless I think there is very cogent evidence of a different kind to show that such a discharge had taken place, and that all the higher centres were for the time being *hors de combat*. It is quite true that there had been no convulsion representing discharge of the cerebral centres; but for hours and days before the convulsion, which I suppose to have been due to discharge in the pons, there was a gradual and progressive loss of all the functions of the higher centres. There was hebetude gradually deepening into unconsciousness; there was languor gradually increasing into helplessness; there was weakness gradually increasing into paralysis; and, perhaps most significant of all, there was that indescribable feeling of horrible depression which, as we see in melancholia, accompanies extreme feebleness of the cerebral energising. All these occurrences point with converging indication to a gradual obliteration of the cerebral functions, progressing from the highest downwards; and it is difficult to see what possible origin this obliteration could have, other than a gradual discharge. Yet there was no convulsion to indicate such discharge. Guarding myself against too dogmatic an interpretation, I would ask if this absence of convulsion may not fairly be ascribed to the extremely deliberate and gradual escape of the liberated energy? That it was very deliberate is shown by the fact that it took many

hours and even days to reach the same degree that is reached in an ordinary epileptic fit in a few seconds. That it was very gradual every observation showed, for no sign of definite stages was ever observed. No fact in nerve-muscle physiology is better established than that the contraction of a muscle depends primarily on the suddenness with which the stimulus is applied to the nerve. A tap, a cut, a shock of electricity—any stimulus whatever that is suddenly or rapidly applied to the nerve, brings about a contraction of the muscle. But a pressure very deliberately applied and very gradually increasing may be added, until the nerve is crushed to pulp, without inducing the smallest movement of the muscle. A continuous galvanic current may pass through the nerve for hours and days without inducing a contraction, save when it is applied and removed; and further, although an increase in the strength of the current is at once followed by a contraction, yet if the increase be very cautiously and gradually made, the contraction may be avoided. It does not, therefore, appear to be claiming too much to suppose that the whole available surplus of energy in the cerebral centres may ooze gradually away without giving rise to any convulsive phenomena whatever; and may thus leave the lower centres uncontrolled and free to indulge in the most rampant excesses of action.

There is a little circumstance which appears to me to corroborate this view. In ordinary cerebral convulsions of deliberate onset we can often cut short the fit by administering chloroform at an early stage. Chloroform, as we know, ablates the function—renders impossible the discharge—of the cerebral centres in their order from above downwards; and if we can thus arrest a discharge, we can of course arrest its extension. But if, as supposed, the convulsion in this case occurred from the excessive discharge of low grade centres, due to removal of the control of the centres above them, then anything that accelerated the removal of this control should accelerate the onset of the paroxysm; and this unquestionably was so. On one occasion, while she was in the stage of "warning," the patient's complaints of her horrible feelings were so bitter, and her sufferings were evidently so severe, that I attempted to give her relief by the administration of chloroform, hoping

thus not only to relieve her suffering but perhaps to ward off the fit. To my surprise, she had no sooner taken a few whiffs than the convulsion began. I say to my surprise, for the very fact that she was able to complain showed that she had not yet nearly reached the stage at which convulsion usually occurred.

The importance of this view here advocated is this, that if it be adopted, it may lead to the explanation of those numerous cases in which maniacal attacks precede the fits in cases of epilepsy,—cases which have always been obstacles in the way of adopting the hypothesis of Loss of Control. In this very case the hebetude and languor that unquestionably indicate a decline of cerebral energising, be the cause of that decline what it may, are not the first occurrences in the attack. They are commonly preceded, as was stated in the notes, I may say, long before this explanation occurred to me, by a period in which her character and disposition are altered. She becomes irritable, quarrelsome, insulting and foul-mouthed; qualities so alien from her usual conduct, that a lady who has frequently visited her, but not at these times, positively refused to believe that she behaved so. She is not actually violent; but this I believe to be simply because she is bedridden, on account of her paralysis, and therefore physically incapacitated from violent conduct; but she uses horrible threats, and this is incipient violence. On one occasion she was acutely maniacal, but this was in the interval between two batches of fits, and may be looked on as post-epileptic as truly as pre-epileptic. Leaving this attack out of consideration, there remains the fact that the very first indication of an approaching fit is a degradation of the disposition, a defect of self-control, and a heightened action of the lower and more brutal nature, similar to that which is common in drunkenness, and which is generally understood to indicate the loss of the highest of all the nervous arrangements.

Not only will the proposed explanation account for pre-epileptic mania, but it may also indicate a relationship between the insanity that occurs in epileptics and insanity that is not manifestly so associated. For if we imagine the progressive degradation that occurred during the "warning"

of the paroxysms to be arrested at a very early stage,—if we imagine it to go no further than that period when her disposition was so changed by what, following Dr. Hughlings Jackson, I suppose to be the emerging into undue prominence of her lower nature, consequent on the loss by epileptic discharge of the highest of all nervous arrangements,—and if we suppose the periodicity of the attack to be unchanged, the case will be one of recurrent mania, and no one would dream of referring it to an epileptic origin. Imagine the process to have gone a little further, and to be arrested at the stage of languor, listlessness, hebetude and intense mental depression,—depression so great that on more than one occasion she has attempted suicide,—imagine this condition to endure instead of sinking to a lower degree, and the case is one of acute melancholia. Let me not be understood to mean that every case of melancholia, or of recurrent mania, is of epileptic origin; all that is contended for is, that when stages in a malady are found to correspond with permanent states existing elsewhere, we are justified in supposing that the conditions underlying these stages are similar to the conditions underlying the permanent states; and that when we know something of the first set of conditions and nothing of the second, it is a tangible advantage to be able to assimilate the two.

There are abundance of other points of interest in this case that might be treated of, did space permit, such as the interparoxysmal spasm of the mouth, its rhythmical character and association with respiration, and the universal tremor that occurred on one occasion, occurrences that might all be shown to harmonise with the explanation offered; but I trust enough has been said, not indeed to secure acceptance of the views proposed, but, what is far more important, to show the great value and importance of systematically recording the phenomena of convulsion. To this endeavour we have in photography an aid whose importance can scarcely be exaggerated. To write down with any approach to completeness the successive phases of a convulsion as they pass before the eye is impossible, and even dictation to a shorthand writer is a very imperfect way of getting a record, so great are the difficulties both of observation and description. But when twenty-four successive

attitudes of a galloping horse can be photographed within the limits of a single stride, there ought to be no difficulty in obtaining a satisfactory record of the successive phases of a convulsion; and now that dry-plate photography is so easy and so cheap, an apparatus ought surely to form part of the armamentarium of every hospital.

ON A METHOD OF DEMONSTRATING THE CONNECTIONS OF THE BRAIN IN HEALTH AND DISEASE.

BY D. J. HAMILTON, M.B., F.R.C.S.E., F.R.S.E.

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ONE of the great wants that must have been felt by those who have worked at the anatomy, normal and pathological, of the brain is that we do not possess any ready means of preparing the organ so as to show the course of the fibres, and their relationship to the different grey centres. We have had plenty of mere microscopic histology, but as to the more remote connections of one part of the organ with another, we know as yet comparatively little with accuracy. In order to see these, it is necessary to examine the brain in thin *entire* sections cut in different planes; for when one part is separated from the other, the various tracts of fibres are interrupted in their continuity, and the very information which we most desire to have fails us. I have now for many years been working on this subject, and have at last arrived at a process by which not only the brain may be cut in its entirety in any direction, but by which the fibres are rendered almost diagrammatically evident. The results which I have obtained by the process I hope to publish shortly, but, in the meantime, as it may be acceptable to those engaged in what is one of the most interesting branches of pathology, I beg to describe the process which I employ.

Hardening of the Brain.—It is of no use trying to investigate the course of the brain fibres unless the organ has been hardened completely into the centre. Any softening which may take place in the process of hardening, from the hardening

fluid not finding free entrance to the central parts, entirely unfits it for any further manipulation. The employment of spirit for the purpose of hardening cannot be recommended, as it causes shrinking and produces consequent deformity. The hardening solution which I employ is a mixture of

Müller's Fluid, 3 parts.
Spirit. Vini Meth., 1 part.

These are mixed, filtered through linen, and allowed to cool before being used. The mixture should never be made until it is going to be employed, and should be kept in the dark.

The brain must be injected with this on three successive days from one vertebral and the two carotids. Unless it is injected, and thoroughly injected, it will never harden. Between fifty and sixty ounces should be injected on the first day, and from thirty to forty on the second and third days. The injection should be driven in with a large syringe, and considerable pressure is required to enable it to penetrate to deep parts. The organ swells to quite double its original bulk when the proper quantity has been forced in. The membranes are sometimes so stretched that they rupture, and the brain may become torn. This must be avoided by not driving in too much liquid at a time. The great danger, however, in injecting is that the points of the canulæ are accidentally driven through the coats of the vessel. This can be avoided by attaching a pliable india-rubber tube about a foot long to each canula, so that the motion of the hand or syringe does not act so directly on its point. When one tube is injected it can be tied up to some support, and thus any tearing of the brain substance by the cross-bar or other part of the apparatus is avoided. The utmost care must be taken in this matter, as a rupture of the internal carotid renders the difficulty of thoroughly injecting the organ much greater.

It is not necessary to inject it while in the body; it injects much more fully when removed. It must, however, be removed with caution, as any tearing, of course, interferes with its perfect distension. The internal carotids should be cut off as long as possible. In removing the calvaria, the outer table should alone be sawn through, the inner plate being broken

with a chisel and mallet. It should be kept in a large round earthenware jar, filled with the same mixture as that which was injected. No padding is necessary—it tends to cause deformity. The best result is obtained by simply having an abundant excess of hardening fluid, and allowing the brain to lie freely in it. The natural shape of the organ is thus preserved.

In a month from the last injection—that is, from the third day after removal—it can be taken out of the fluid and cut into entire slices one inch thick with a long thin knife, in the particular direction in which it is intended to make the sections. These should lie in the fluid for another fortnight. If the injection has run properly, the parts internally should be tough and thoroughly impregnated with the Müller's fluid mixture. The great test of the injection having been successful is that the septum lucidum and surrounding parts are entire and perfectly hardened. The hardening has now to be completed by placing the segments in pure methylated spirit. This can be done without shrinking occurring when the brain tissue is fixed by means of the former fluid. I generally allow it to lie for two weeks in the spirit, and care should be taken that the segments are separated from each other by pieces of linen or cotton-cloth, or some other absorbent substance.

Colouring of the Convolutions.—When thin sections are made of the brain, it is almost impossible to tell what convolutions are immediately under observation. In order to mark these out, it is necessary to colour them differently with some material which will adhere to the pia mater or arachnoid membrane. The mixture which I have found most serviceable is gold-size and different ordinary pigments in powder. They are to be ground up in a mortar together, and applied by means of a camel's-hair brush, the colouring matter being well rubbed into the membranes, so as to take a firm hold. The best stage for putting the colour on is when the segments are removed from the methylated spirit. They should be all re-adjusted, so as to see exactly where the convolutions lie, and should be partially dried outside by exposure for two or three hours before being painted. A good thick layer of the paint is necessary in order to be seen in a thin section. The colours used should be such as are not decomposed by potash. Potash

has to be employed later on, and certain colours fade by its action. The best that I have found are vermilion, lamp-black, and oxide of zinc. They should be applied individually to convolutions as far apart as possible; and by leaving some uncoloured and by colouring others, there can be no uncertainty as to the part of the cortex concerned. A key to the order in which they are coloured should be kept for future reference, and it is well to colour all brains alike to prevent confusion. One side only should be coloured, and always the same side. I generally employ the right. The particular side of the brain is thus indicated, whatever the surface may be that is uppermost. It is a very good method to follow in the investigation of the spinal cord as well.

Preparation of the Brain for Cutting.—The only method by which the sections I am about to describe can be cut is by freezing. Any one who has seen the ease with which it can be done in a frozen brain, and the difficulty—or, I should rather say, the impossibility—of cutting a regular series of sections by any other method, will be convinced of this. The brain, however, must be specially prepared for freezing, as otherwise one might as well try to cut through a piece of rock, so hard does it become. Unless specially prepared, its structure will also be so damaged by the pressure of the ice crystals as to be totally useless for my method of bringing out the fibres. It must be impregnated with some mixture which will prevent it from freezing too hard. In a paper which I formerly published on the *technique* of this subject ("Journal of Anatomy and Physiology," Vol. XII.), I recommended soaking the brain tissue in simple syrup of a certain strength. I have since found out that the following is much preferable:—

	ozs.
Syrup (1 oz. by weight crystallized sugar to 1 fl. oz. of water)	4
Mucilage (1 lb. gum acacia to 80 oz. water.)	5
Water	9

Mix and saturate, by boiling, with boracic acid.

Filter through muslin.

The above strength is the only one which will serve the purpose. I have experimented with many other proportions, and have found by experience that this on freezing, after

having soaked into the brain tissue, produces the best consistence for cutting.

The method of employing this freezing mixture, is firstly to get rid of the spirit by soaking the segments in water which is constantly renewed for at least three days. It is usually better to allow from four to five days, according to the thickness of the segments. They are afterwards immersed in a large quantity of the freezing fluid, so that they may be thoroughly soaked by it, each segment being separated from its neighbour as before described.

They should lie in the freezing fluid for a fortnight at least, and the longer they are immersed in it the better. I keep them permanently in this until they are wanted.

Cutting of the Sections.—The segments are now ready for cutting. The microtome which I have found to be most serviceable is one made for me by Mr. Gardiner, surgical instrument maker, Edinburgh. It is extremely simple in construction, and the freezing part of it is essentially on the same principle as that described by Professor Rutherford, there being certain necessary additions in regard to the knife. The well is nine inches in diameter, and a very powerful finely graduated screw moves a cylinder upwards on which is placed the piece of brain to be cut. The surface to which it has to be frozen is roughened by means of a series of concentric rings, so as to prevent the piece of brain from becoming detached. I fix the piece of brain on *with the freezing mixture* just described. Mucilage does not suit, as it freezes too hard and becomes brittle. The section is apt to tear in being detached in front, unless there is some mass to support it. I usually fill the well here with the freezing fluid, but at other parts I leave it unfilled, simply freezing the brain on to the top of the grooved brass cylinder.

The best freezing mixture is salt and snow, and it must be pressed in all round the well, and stirred from time to time. It freezes much more quickly and more equally if the water resulting from the melting of the snow is not allowed to run off until it becomes excessive. The quantity of snow required varies of course with the temperature, but I have found that in moderately cool weather half-a-hundred weight is more than

sufficient to keep the machine frozen for a day. Ample time is thus afforded for preserving every section from a piece of brain of the thickness formerly indicated (1 inch).

The knife has given me much more trouble to adjust than the freezing part of the apparatus. The surface to be cut is so large, and the tissue when frozen so hard, that any ordinary knife constructed similarly to those employed for smaller microtomes is utterly useless. The only cutting instrument at all applicable is one made on the principle of a plane. That which I employ has a framework made of brass, and is very massive, so as to afford a considerable momentum. It is exactly like a large Canadian plane, with the front part of the framework cut out. The blade rests upon three solid metal supports behind, and is fixed to them by a screw. The blade is made of a thick plate of steel, and the whole framework runs in a grooved railway at each side of the plate of the microtome, so that lateral or other deviation is prevented. The edge of the blade being sufficiently strong not to bend, is thus bound to run through the frozen brain segment. The railway entirely does away with the necessity for having a plate-glass top to the microtome, as the blade never comes in contact with the metal in being swept forwards.

In employing this cutting machine, the railway should be well smeared with glycerine, and the blade is driven forwards with great rapidity. It must be perfectly dry, and as cold as possible. I formerly used to freeze the blade with a couple of boxes containing a freezing mixture placed in contact with it behind. The object of having it dry and cold is to prevent the section sticking to it. If this happens the section is ruined, as no amount of manipulation will be sufficient to detach it entire. It ought to be thrown off the blade, to turn once over, and to land on a flat vessel containing water about a foot and a half to two feet in front of the microtome. The beauty with which this can be done by means of this instrument requires to be seen to be appreciated. The section thus lands on the surface of the water before it has thawed, and it continues to float here usually perfectly spread out, or, if not so, capable of being unfolded by means of a couple of camel's-hair brushes. The section is so fragile and so large,

that unless it were thrown off in this way I cannot see how it would be possible to prevent destroying it. Each section must be removed from the water separately as it is, cut on a glass plate prepared in the manner now to be described.

Method of Fixing the Section on a Glass Plate.—As just mentioned, the section is thrown from the blade of the cutting machine on to water contained in a flat dish in front of the microtome. Previous, however, to this, a plate of common window glass (12 × 11 in. is the best size), is covered with a layer of French gelatine, and placed in the flat dish. The gelatine solution for gelatinising the plate should be made in the following manner:—Soak a quantity of French gelatine (Coignet's is best) in water for half-an-hour; melt this in a water-bath, and measure the quantity of the solution. Take three times as much water, and add to it sufficient carbolic acid to make the whole mass up to a 1-to-40 mixture; gradually add the water with the dissolved carbolic acid to the warm gelatine solution. Pour it over the glass plate with a pipette, so as to make a pretty thick uniform layer.

This gelatinising of the glass plate ought to be done immediately before the section is cut. The gelatine should be allowed to firm, but not to dry, a few minutes after it is applied being sufficient for the purpose. If placed in water, numbers of air-bells will be found to adhere to the surface, but these can be easily removed by holding the plate under a gentle stream of water, and lightly rubbing the gelatinised surface with the finger. This gelatinised glass plate is to be the recipient of the section when it is thrown on the surface of the water; and as a great commotion of the water and consequent disturbance would result if it were put into water after the section had been cut, it is necessary to do so as a preliminary measure, and to allow any disturbance of the water to subside.

There should not be too much water in the flat vessel. About half-an-inch is the most that is required. If more than this is employed, there is difficulty in getting the section out on the slide. The glass plate is gradually lifted up out of the water, bearing with it the section which is floating on the surface. This slide is not to be the one on which the prepara-

tion is to be permanently mounted. Hence it is immaterial in what position it lies upon it. This is a very great advantage, as it is extremely difficult to fix it on a particular part of the plate while in its present unstable condition. The water running off from the plate tends to displace it. Whatever position it is in when removed, this should be retained, and no attempt made to alter it by means of brushes or otherwise.

The gelatinised slide, with the preparation upon it, is now placed in a gently sloping position. About half-an-inch elevation is requisite, so as to allow the water to drain away, and, at the same time, prevent the preparation sliding off the plate. This position should be maintained until the superfluous water has been entirely got rid off, but the preparation must not be allowed to dry. If any air-bells are enclosed under it, they can be drawn to the side with a damp camel's-hair brush, or dissipated by touching them with a hot needle.

The preparation at present has a layer of gelatine under it. A second layer of the same gelatine solution is now dropped over it with a fine pipette. It should be put on at a high temperature, and the aperture of the pipette should not be too large, as a gush of gelatine solution is liable to occur, which may destroy the preparation. This is allowed to cool, and now the preparation is perfectly enclosed between the two layers. These become inseparably united, and it is thus fixed upon the slide. It should be labelled at once by some distinctive symbol and number indicating its position in the series.

Method of Defining the Fibres and Enlarging the Preparation.—As looked at in this stage, one would hardly believe that the preparation is the same as it turns out to be when finished. It is perfectly opaque, and the definition of the white from the grey matter is very unsatisfactory. In order to give a demonstrative view of the fibres, it should not be extremely thin—not so thin as one usually cuts brain for ordinary microscopic work. Consequently, were the preparation retained in this state permanently, it would be of comparatively little use for the object I have in view. Some means must be adopted for clarifying it, and for bringing the bundles of fibres into prominent contrast with the grey matter. For this purpose

the usual clarifying agents, such as oil of cloves, turpentine, or glycerine, are quite inapplicable. The first and second clarify too uniformly, and do not differentiate the white matter from the grey sufficiently well. The whole preparation becomes uniformly transparent. After trying many different ways of accomplishing this, I have now discovered a method which, for the purpose of demonstrating the white tracts and the grey nuclei for naked-eye purposes is, I believe, better than anything we possess. It is the following :—

The preparation, covered above and below with gelatine solution, and in its yet opaque condition, is allowed to dry. Several days' exposure is usually necessary for this end. When it dries, it becomes perfectly transparent. The gelatine prevents it from cracking or shrinking. It is converted into a thin transparent film, firmly adherent to the glass plate. The gelatine becomes thoroughly incorporated with it, so that the textures of each are practically continuous. The differentiation now is even worse than before, because the white and grey matters are equally clarified.

The dried preparation, and the gelatine for about two inches round it, are next brushed over with liquor potassæ (British Pharmacopœia). Sufficient is required to soak it thoroughly, and it should be spread over the surface with a brush, so as to equally distribute it. It is allowed to remain in contact with the preparation for five minutes, and the slide is then elevated at one end, so as to allow the superfluous potash to run off. If any gathers at the lower part of the slide, it should be brushed away.

The preparation so treated is now removed to a cool place for twenty-four hours, and if at the end of this time the gelatine be cut round at about a distance of one inch from its edge, the whole of the preparation incorporated with a film of gelatine can be stripped off from the surface of the glass. If it is not perfectly dry, it should be laid on the reverse side of the glass plate until it is so. The preparation in this state can be handled with impunity, just as a sheet of dry gelatine can. The two have become so incorporated that they appear to be one tissue. The preparations should now be labelled with black varnish, gathered into bundles, and reserved for further treat-

ment. They keep, if retained in a dry atmosphere, apparently for any length of time, and can be sent by post, or be otherwise conveniently disposed of, as the case may be.

I discovered in working at this subject that if gelatine be dried, and treated with potash as described, it becomes so hygroscopic that when placed in water a sheet of it will expand to nearly double its original size. If glycerine is mixed with it the same swelling occurs, but to a minor extent; and I further made out that if a section of brain is enclosed, as mentioned, in the film of gelatine, it also expands to a similar degree, and, at the same time, there is produced the most beautiful differentiation of the white from the grey matter. If one of the above-described gelatine films, with the enclosed preparation, be placed in water for half-an-hour, it will be found to have grown from one half to double its original size. The grey matter remains quite transparent, while every bundle of fibres in the white matter is marked out with the utmost clearness. The beauty of the preparation in this stage is very striking, especially as contrasted with its appearance before being immersed in the water.

It can even yet be handled without much fear of injury, and can be turned round in the water, so as to get rid of any underlying air. A glass plate, similar to the one before described, is next slipped under it while in the water, and the preparation withdrawn. There is now no difficulty in adjusting it in proper position on the slide, as the casing of gelatine protects it. It might be supposed that the consistence of the gelatine would be destroyed by the action of the potash. This is not so if too much is not employed, and if it be applied only once. If there should be any difficulty in getting the preparation off the slide after the potash has been added, it is usually due to its having dried too much during the twenty-four hours which have elapsed from the time of application of the potash. If so, all that is necessary is to damp the adherent part slightly. It usually strips off without any difficulty, the potash having the effect of loosening the hold of the gelatine upon the surface of the glass.

By the method just described, a two-fold object is gained—namely, that all the parts of the preperation are much enlarged,

and the white matter is perfectly differentiated from the grey. The ratio of expansion seems to be equal throughout; so that every part, although enlarged, presents the same relative size to others. The advantage of this method of enlargement is that it makes small objects more conspicuous, and at the same time, by separating the fibres, dissects them out to a certain extent, and renders the tracing of them much easier. By repeating the process, and again expanding, a still further enlargement follows; so that one can go on successively enlarging in this manner until an enormous preparation is produced. I do not know how large such preparations could be made, but I have some in my possession which are more than twice their original superficial area.

At each fresh application of potash and gelatine, the differentiation also becomes more pronounced. Nor is the preparation rendered useless for microscopic purposes. The fibres can be traced perfectly, either with a low or high power, if such should be considered necessary. By far the most instructive picture, however, is gained by unaided vision, or by the use of a simple lens of large size.

The Mounting of the Preparation.—The section having been expanded and taken out of the water on a glass plate, the superfluous water should now be allowed to run off. This takes about twelve to twenty-four hours, and it should not be further treated in any way until after this time.

It must be mounted in glycerine jelly, made in the following manner:—Soak French gelatine in water as before until it is saturated, melt it, and measure out equal quantities of German glycerine and of water, adding to them sufficient carbolic acid to form a 1-to-40 mixture. Add the water, glycerine, and carbolic acid to the gelatine in a water-bath.

A layer of this glycerine jelly must be put over the preparation and allowed to harden before the cover-glass is adjusted. The reason of this is, that in being covered it is liable to become displaced unless fixed to the surface of the glass. There are other reasons which render this procedure necessary. The glycerine jelly is dropped on the preparation from a pipette at a temperature of between 80° and 90° F. If higher than this, it will melt the gelatine around the preparation and destroy it.

A nice even surface is thus formed, over which a cover-glass can be easily slipped. If there should be any opaque parts in the preparation, it should be left in this uncovered state until they disappear. It is usually advisable to allow it to remain uncovered for three or four days under any circumstances. It will then be ready for mounting. This is accomplished by first holding it under a gentle stream of water for a minute or so, and rubbing it softly with the hand until the surface is thoroughly wet. The same glycerine jelly as before described is now placed over it, at the formerly-mentioned temperature, and the cover-glass, previously warmed, is slipped over this. In a very short time the glycerine jelly fixes, and then the preparation is safe.

The superfluous glycerine jelly outside ought during the same day to be removed, the surface washed with hot water, and the edges of the preparation cemented with the following :—

Gum Damar 8 oz.
Benzole 8 fl. oz.

Let them dissolve. Filter, and rub in with a pestle and mortar 7 drs. of oxide of zinc. Filter through muslin. The greatest trouble may be caused by air getting in at the edges, unless the cementing is done on the same day as the mounting of the preparation. The cover, it may be stated, is made of common window glass, similar to the slide on which the preparation is mounted. It should always freely overlap the edge of the preparation.

The process which I have described is specially applicable for large brain sections. It might, however, be employed for other tissues where it is desirable to keep the parts *in situ*. I should think that sections of the eye-ball would be specially demonstrative if mounted in this way.

ON LOSS OF BOTH KNEE-JERKS FROM ONE-SIDED BRAIN DISEASE.

BY STEPHEN MACKENZIE, M.D., F.R.C.P.

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I PROPOSE to discuss a very interesting phenomenon, which offers considerable difficulty of explanation, namely, the loss of both knee-jerks from a unilateral cerebral lesion. It will, I think, be found that the condition is not by any means of rare occurrence, but attention has, as far as I know, scarcely been called to it, and I am not aware of any explanation that has been advanced concerning it. It will perhaps be most convenient if I first relate a case in which the phenomenon existed. It was a case of gliomatous tumour of one hemisphere causing hemiplegia and hemianasthesia, whilst the patient had complete loss of both knee-jerks. The case is as follows:—

M. N., aged 25, came to the Ophthalmic department of the London Hospital for defect of sight, on the 8th of January, 1880. My colleague, Mr. Waren Tay, under whose care she came, noted "Myopia; changes in yellow spot region (resulting from hæmorrhage?)

L. $3\frac{1}{2}$ Snellen $\frac{20}{0}$ — 3 D $\frac{20}{70}$

R. $3\frac{1}{2}$ Snellin — 11 D $\frac{20}{100}$

Evidences of neuritis, with hæmorrhages, in *left eye.*" On the 15th of March, when seen again, Mr. Tay noted: "The *right* disc is now distinctly swollen, the margins obscure, and there also appear to be small nodules of lymph here and there. In addition, of course, are the myopic patches of more or less exposed sclerotic. No hæmorrhages detected in right eye. In left eye, well marked swollen disc, with lymph and hæmorrhages."

The following history of the case was elicited by Mr. Tay and Mr. Makeham, then my clinical clerk:

"Family history unimportant. The patient was backward in teething and walking, and at school, in learning. She had a discharge from her ears, accompanied by pain, from the age of three until dentition was completed. When 9 years of age had disease of right hip, following scarlet fever, which left her permanently lame. Since the age of 15 has been engaged as a milliner.

"Just before Christmas, 1879, she complained of pains in the head, and of a cold numb feeling down her right side. On the 7th of February, 1880, she appeared to lose her sight almost entirely and somewhat suddenly, but she had complained of defect of vision since the beginning of the year, and as already stated she was seen by Mr. Tay on the 24th of January, and her condition then noted. At the time when the loss of sight became nearly complete she had very severe frontal headache. One week before admission she had two fits, with an interval of fifteen minutes between them. In the fits she was unconscious, and had convulsive movements almost confined to the right side. When she came out of the fit she squinted a little, temporarily. Since the fits she has not been able to walk, has seemed very drowsy, and has slept a good deal. Mr. Tay, recognising the nature of the case, asked me to see the patient on the 20th of March, when I made the following note:

"The right hand and arm are feeble. She drags the right leg in a feeble way, and has not power to stand on it. She cannot stand or walk at all unassisted. The pain in the head is very severe, and only relieved temporarily by hypodermic injections of morphia. There are occasional movements of right eye, inwards and a little downwards, independently of the movements of the left eye. These movements are increased when she looks to the left. The external rectus of each eye appears feeble. The double optic neuritis, severe pain in head, vomiting, and slow onset of paralysis, all point to tumour of the brain."

The patient was transferred by Mr. Tay to my care on the 20th of March, when it was noted as follows: the eyeballs

are prominent, the corneo-sclerotic junction being seen all round when she opens her eyes. Can just see moving objects. Tongue clean, bowels much confined. No abnormal physical signs in chest.

As already mentioned there is decided loss of power in the right arm and leg, but no facial paralysis or deflection of the tongue are detected. *There is entire absence of both knee-jerks.*

April 1st.—Yesterday afternoon facial paralysis was noticed by the sister of the ward. To-day the mouth is slightly drawn to the left. When she parts her lips the mouth is much more arched on the left side. The right eye closes imperfectly upon making an effort. The left eye winks naturally, whilst the right scarcely moves. When she frowns no wrinkles can be seen on the right side. The tongue on protrusion is slightly deflected and curved to the right. Uvula not deflected. Sensation to heat, cold, pain, and ordinary tactile impressions are impaired on the right side. She cannot move the right lower extremity. *There is complete loss of both knee-jerks.* No increase of plantar reflex on right side. She cannot walk now, even with assistance.

April 3rd.—Complains of constant headache. No vomiting. Facial paralysis still more marked. Cannot hear a watch close up to right ear. Smell absent. Taste normal.

April 6th.—Since the 2nd of April has not been able to distinguish night from day.

April 9th.—I noted the paralysis of motion of right face, arm, and leg is very complete. Owing to the patient's blindness, it is difficult to test the ocular movements, and all that can be said is that the right superior rectus does not move so freely as its fellow. There is marked, but not complete, loss of sensation on the right side of face, and the sensation of the right arm and leg is very much impaired, being only observed on deep pricking. She can now hear a watch with right ear at about one inch distant, on the left side at four inches distant. There has been no incontinence of urine or fæces.

April 12th.—Facial paralysis less marked. Can feel better the prick of a pin. She has also regained to a very slight degree the movement of both upper and lower right extremities.

April 19*th*.—Complains very much of pain in the head. Has vomited several times the last few days.

April 20*th*.—The minute vascular injection of the left disc is less conspicuous, the tissues being more opaque. The retinal veins are very large and tortuous, the arteries small, and there are a number of hæmorrhages on and around the disc. Between the disc and the yellow spot are a number of white striæ converging to the latter point. Her urine contained a trace of albumen on admission, none since.

April 23*rd*.—The left eye appears slightly more prominent than the right. The palpebral fissure is smaller on the right side, apparently owing to want of tone, for she can both open and close the eyelids, but she closes the right more freely than the left. The movements of the eyes are limited in all directions, especially upwards.

April 28*th*.—Has become more and more helpless, the pain in the head has been very severe lately.

April 29*th*.—Swelling of right upper lid, conjunctiva injected, and one or two abrasions over cornea. The whole of right side of face is injected, and there is a profuse mucous discharge from the right nostril.

May 2*nd*.—Patient complains of pain in the *left* extremities—"As if they are being pulled away from me," she says. She is losing power on the left side, this has been noticed for some days. She cannot draw up the left leg; cannot lift the left arm from the bedclothes; the grasp of the hand is very feeble. No anæsthesia on left side.

May 6*th*.—Suffocative attack, associated with a choking cough, in which she became livid in the face.

May 9*th*.—After a trifling improvement is again worse. She has had another "choking fit" to-day, this being the third. They come on after drinking something, and nurse says, "she seems as if she had not sense enough to swallow properly." Following this last attack she sank into an unconscious condition, the heart's action became irregular and intermittent, the respiration feeble and gasping, and she died at 10.10 P.M.

The *necropsy* was made on the following day. The detailed description of what was found is mislaid, but the following

transcript from the abridged *post mortem* records supplies all that is essential to be conveyed:

"Glioma of left hemisphere (with hæmorrhage) involving left corpus striatum and thalamus opticus, and extending down crus cerebri to its junction with the pons. No changes in viscera except venous congestion."

The spinal cord showed no changes to the naked eye. It was hardened in bichromate of ammonia, and has since been examined. Sections from cervical, dorsal, and lumbar regions show no descending wasting, and no changes of any importance.

There are several points of interest in this case, to which I may in passing allude.

I. That the symptoms, though not strictly beginning in the eye, so far predominated there that the patient first came under the care of the ophthalmic surgeon. When first seen by Mr. Tay there was unilateral neuritis. It was the left eye which was first affected with inflammation and the disease causing it was also on the left side of the brain. As I have shown [1] when optic neuritis is unilateral it has usually been on the side opposite to the growth. Dr. Ernest Field [2] has also recorded a case illustrating this fact. But whilst I think this is often the case, it is clearly not, and I did not in drawing attention to the point intend to imply that it was, invariable. As regards the ocular movements and paresis, but little can be said, but the tumour involving the thalamus was in close propinquity to the centre discovered by Adamuk in the corpora quadrigemina and thus may be explained the movements and weakness of the muscles of the right eyeball from disease on the left side of the brain, no disease being found on the right side. Owing to the patient's state these conditions could not be very accurately studied.

II. The symptoms enabled a localisation of the disease to be made with certainty. The dysæsthesia followed by anæsthesia pointed to the thalamus opticus or its vicinity: the right sided convulsion followed by gradual and extending paralysis of motion of the right half of body, pointed, in conjunction with the anæsthesia of the same area to disease involving the

[1] 'Brain,' vol. ii., p. 269. [2] Ibid. vol. iv., p. 249.

corpus striatum, or anterior part of internal capsule. The severe headache, vomiting and double optic neuritis, made it certain that the local disease was tumour. The absence of syphilis, acquired or congenital, the absence of signs of decided struma, and the age of the patient rendered it probable that the tumour was, as it proved to be, gliomatous.

III. The hæmorrhage into the tumour just before death. I regret that my detailed notes on this point are not available. There was a large black clot in the growth. We know that this not infrequently happens in vascular gliomata, that it may give rise to apoplectiform symptoms, and in the inexperienced, especially in the absence of history, lead to the growth which has bled being overlooked. This would be all the more likely when, as in this case, the growth occupied "the seat of election" of hæmorrhage.

The point to which I especially wish to draw attention in this case, however, is not any of the foregoing, but to the double loss of knee-jerk. It will be observed the cerebral disease was strictly unilateral, the loss of knee reflex double. I believe in this respect this case is by no means an isolated one. I have seen more than one case and know of others in which there has been clinical evidence of cerebral tumour with double loss of knee-jerks but in which there was no post mortem. Dr. Hughlings Jackson mentioned at a discussion at the Ophthalmological Society recently a case in which there was local brain disease, but in which no change was found in the cord after death [1] and since then I have seen the parts from a case of his in which there was unilateral tumour loss of both knee-jerks, and no naked eye changes in the cord at the necropsy. Dr. Hughlings Jackson alluded to other cases he had seen without necropsis.

The *loss* of knee-jerks from cerebral disease is difficult of explanation. We know that the brain exerts a controlling or inhibitory power over the spinal cord, as regards its reflex functions; and that this inhibition can be increased by a voluntary effort. The exact course of the inhibitory fibres in the brain and cord is not certain, though as regards the latter it is probable that the fibres pass along the pyramidal tracts.

[1] 'Lancet,' June 15, p. 1046.

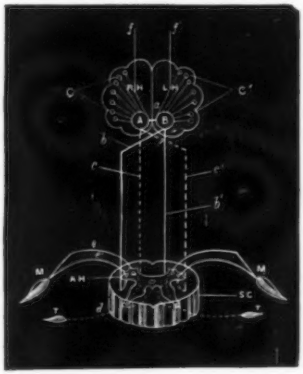
It is well known that when the cerebral inhibition is removed, as by a destructive lesion between the medulla and the level of the cord, concerned, as a centre, in completing the reflex, exaggerated reflex is the result. How then is it to be explained that a cerebral lesion causes abolition of the reflex?

I venture on the following explanation with the greatest diffidence, and my putting it forward may have the effect of eliciting a better one. Suppose there is an inhibitory centre (or centres) in each hemisphere acting upon the spinal cord as a reflex centre. These centres under ordinary circumstances act together, and control the reflex excitability of the cord. Their inhibitory power is capable of being augmented by a voluntary effort, say by means of cortical centres connected by commissural fibres with the inhibitory centres lower down.[1] The two inhibitory centres naturally act together, each being also connected with both halves of the spinal segment concerned in the reflex, in the same way as motor impulses are conveyed to both halves of the cord by the crossed and direct pyramidal tracts. But being arranged for combined action, if one is injured or destroyed, the other is uncontrolled; and connected as it is with both halves of the cord, over-inhibits or annuls the reflex affected through both halves of the spinal segment concerned. The injured, or destroyed, normally cooperating inhibitory centre necessarily has its function abolished; and, if there were no other action, this would lead to exaggeration of the reflex governed by the inhibitory centre. But this is prevented by the uncontrolled healthy inhibitory centre whose positive action outweighs the negative action of its destroyed fellow centre. In this way can be explained how a lesion limited to one side of the brain can cause loss of both knee-jerks, and this without there being any structural alteration in the spinal cord. The diagram on page 231 will, I hope, serve to illustrate my meaning.

Let us suppose A and B, connected by the commissural fibre

[1] This is certainly less probable as regards the knee-jerk than as regards the vesical and some other reflexes. I am aware of the powerful arguments against the knee-jerk being a true spinal reflex, but it is enough for the argument to admit, as is universally done, that it depends upon a spinal influence.

a, to be inhibitory centres in each half of the brain (R H and L H), A and B are connected with higher centres C and C′, by which their inhibitory power can be strengthened. From A



R. H., right hemisphere of brain. L. H. left hemisphere. A. right cerebral inhibitory centre; B. left ditto, ditto. C. cortical centres. A. H. ganglion cells of anterior horn of (S. C.) spinal cord. M. muscles moving knee. T. patellar tendon. *a*. commissural fibres connecting cerebral inhibitory centres: *b* and *b*^{1}, paths of cerebral inhibitory fibres from left half of brain to both halves of cord: *c* and *c*^{1}, paths of cerebral inhibitory fibres from right half of brain to both halves of cord. *d* sensory fibres from patellar tendon to grey matter of cord: *e* and *e*^{1}, motor fibres from anterior horn to extensor muscles moving knee: *f* and *f*^{1}, fibres from cortex to inhibitory centre.

fibres pass by C and C′ to the right and left of the spinal segment (S C), where, in the anterior horns (A H), they communicate with the ganglionic centres there situated, and exercise their inhibitory function. From B fibres pass by *b* and *b*′ to the spinal segment in similar manner. T represents the patellar tendon (I assume, for simplicity of argument, that the knee-jerk is a tendon reflex), and M the extensor muscles moving the leg. A stimulus from T is conveyed along *d* by the posterior root to the spinal cord, and an efferent impulse sent by the anterior horn along *e* to the muscles (M). In ordinary circumstances the reflex completed in the spinal segment, say in the anterior horn, is here controlled by the fibres, say from the crossed and direct pyramidal tracts, coming from the inhibitory centre of each side of the brain. Destruction of one half of the cord above the segment concerned in the reflex, involving *b* and *e* or *b*′ and *e*′ will lead to exaggerated knee reflex on the injured side, by removing the influence of both cerebral inhibitory centres; and destruction of both halves of the cord above the segment will lead to exaggerated knee reflex on both sides. This is the explanation usually received.

If, however, there be a lesion of one side of the brain invol-

ving this inhibitory centre A, the influence of A is removed from both halves of the cord; but B, being unantagonised, overacts, and being connected with both halves of the cord, *annuls both knee reflexes*. A difficulty immediately suggests itself as to adequacy of this explanation. In hemiplegia, due to a unilateral cerebral lesion, there is usually exaggeration of the knee-jerk on the paralysed side. This is generally said to be caused, but more correctly described as permitted, by the secondary degeneration of the cord above the spinal segment concerned in the reflex. It would appear to me to be due to, or permitted by, involvement of parts below, A and B in *b* and *c*, or *b'* and *c'*, and if so it would not militate against my hypothesis. It will be observed in my case, that though there was right hemiplegia, there was no exaggeration of the knee-reflex on the paralysed side. These corresponded with the absence of descending sclerosis in the spinal cord, and thus harmonises with my hypothesis.[1]

As I have already said, it is not ascertained how the fibres travel to the spinal segment from A and B, or whether they are independent of the motor fibres passing from the brain to the cord. This hypothesis, it will be seen, explains how the knee-reflex is exaggerated when there is descending wasting in the cord, and at the same time explains why there need be no lesion in the cord when the reflex is abolished on both sides from a unilateral cerebral lesion, as in the case I have narrated, and as occurs probably not unfrequently. On my hypothesis, if *both* inhibitory centres were disabled, there would be exaggerated knee-reflex on both sides, from complete removal of all cerebral inhibition, in the same way that a total transverse lesion causes exaggeration of both knee-jerks. I do not know, however, of any facts bearing on this point.[2]

[1] The increase of knee-jerk noticed after convulsive seizures would appear to be due to exhaustion of the hypothetical inhibitory centres.

[2] See a very interesting case of loss of both knee-jerks from tumour of cerebellum, in a case of Dr. Ramskill's, reported by Dr. Coxwell, 'Brit. Med. Journal,' May 19, 1883, p. 957.

ON A CASE OF "SAWYER'S CRAMP."

BY G. V. POORE, M.D., F.R.C.P.

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THE patient whose case is recorded in the following short paper was sent to me by my friend Dr. de Watteville, who was kind enough to remember that I took an interest in "professional" ailments.

W. W., æt. 38, came as an out-patient to University College Hospital on the 8th of March, 1883. He was a maker of packing-cases and had been engaged in piece-work, making his packing-cases against time, as it were, for 10 or 11 hours a day, and occasionally for as much as 14 hours. He was a strongly-built man, but looked a little worn about the face, and had a certain earnestness of manner which I have often seen associated with genuine professional ailments. He did not drink, and there were no facts in his personal or family history which need to be recorded. His only trouble was an inability to use the saw. As soon as he took his saw in his hand, his efforts to use it were thwarted by disorderly movements which he could not explain.

In order to better understand the case, I asked him on the next occasion to bring his saw with him to the hospital, together with a piece of wood such as he habitually used in his trade. This was done, and the patient was stripped to the waist, and was made to go through the act of sawing.

On taking the saw in his hand, there was noticed a slight tremor of the blade, due to a little shake of the hand. The saw was thrust forward through the wood fairly well, but the back stroke could not be accomplished, and the attempt was accompanied by abnormal elevation of the shoulder, scapular movement, adduction of the elbow, rotation outwards of the forearm, flexion of the wrist, and swaying to and fro of the

body. These movements were difficult to analyse, the general impression left upon the observer being that the whole of the muscles of the right upper limb behaved tumultuously when the patient attempted to saw.

With the left arm he could saw perfectly well.

From the character of the movements I thought it not unlikely that some of the scapular muscles were at fault, and my first step in the examination was to outline both scapulæ with a blue pencil, to see if there was any want of symmetry in the position of the scapulæ on the two sides. This examination gave a negative result.

On close inspection, there was seen to be distinct flattening of the right supra-spinous fossa as compared with the left. That this was not due to any wasting of the trapezius was shown by putting the trapezii in action, when the muscles on the two sides appeared to be equally developed. The first positive fact with regard to this patient then was—

(*a.*) Diminutions in size of the right supra-spinatus muscle.

On close inspection of the chest, there was noticed a slight degree of flattening of the right infra-clavicular region, and I came to the conclusion that there was

(*b.*) Diminution in size of the right pectoralis major, especially the clavicular portion.

The muscular wasting in the above situations, though very slight, was undoubted, and when pointed out was readily admitted by all who saw the patient.

I was almost inclined to think that there was a little want of plumpness about the right infra-spinous fossa, and that the muscular mass between the spinal border of the scapula and the spine was more flabby on the right than the left side; but of these two facts I could not feel at all sure. No other muscles except those in the situations named were in the least degree wasted.

The next step in the examination was to determine if any of the nerves of the brachial plexus were tender. There was no tenderness above the clavicle, nor over the situations of the great nerve trunks (musculo-spiral, median, or ulnar) in the upper limb.

(*c.*) There was a markedly tender spot in the right pectoral

region, in the second interspace, midway between the mid-sternal line and the point of the shoulder.

(*d.*) An equally tender spot beneath the spine of the right scapula, just where the supra-scapular nerve enters the infra-spinous fossa of the scapula. The next step was to test the irritability of the muscles to faradism. It was found that the muscles of both upper limbs manifested equal degrees of irritability with one exception.

(*e.*) Both portions of the right pectoralis major (clavicular and sternal) showed a marked degree of excessive irritability, and contracted readily to a current which produced no effect on the left side. The right muscle was far more sensitive to the current, and manifested what Duchenne would have called excessive amount of electro-muscular sensibility.

It must be remembered that the position of the supra-spinatus (which was wasted) prevented its being accurately tested, since the movement of the trapezius effectually masked any movement of the muscle beneath it.

The patient stated that he could perform every movement and every act with the right hand with perfect readiness, with the exception of the act of sawing, which, unfortunately, was the one upon which his livelihood largely depended.

Upon examining the individual movements of the limb with great care, it was found that the approximation of the scapula to the mid-vertical line (by means of the rhomboids) was not so readily accomplished on the right side as on the left; and that when the latissimus dorsi was put into extreme action, he complained of pains in the pectoral region. I am inclined to think that the difficulty of the rhomboidal movement, and the pain during the action of the latissimus dorsi, were both in part attributable to the stretching of the pectoralis major which these movements caused.

Questioned as to the origin of his trouble, he stated that it began in March, 1882, and that the first symptom was a pain in the pectoral region during the back stroke of the saw (during which movement the pectoralis major was certainly put on the stretch).

It will be observed that the case lends no support to the statement which is generally found in text-books, that in pro-

fessional ailments of this kind there is nothing the matter except the derangement of a special co-ordinated movement.

In this patient distinct evidence was obtained of derangement of special muscles and special nerves, but this derangement was not of a kind to attract the attention of a careless observer. On the contrary, it was only discovered after a very diligent search.

This case, again, lends no support to the theory that ailments of this kind are due to the failure of some co-ordinating centre.

Two muscles were found slightly wasted (the supra-spinatus and the pectoralis major), and the nerves supplying them were distinctly tender; at least two sharply defined tender spots were found over the anterior rosacic and the supra-scapular nerves.

It will be at once conceded that an irritable condition of the pectoralis major would be likely to seriously interfere with the act of sawing.

The connection of the supra-spinatus with the act of sawing is, however, not at first apparent.

In my papers on "Writer's Cramp" I have insisted on the fact that the muscles which are subjected to prolonged strain are more likely to be affected than those in which contraction and relaxation quickly alternate, and accordingly we find that the muscles of pen-prehension more often suffer from over-use that the muscles of pen-movement.

In the act of sawing, what muscles, if any, are subjected to prolonged strain? Not the muscles of the hand, for the saw is alternately thrust and pulled, and, during the former act the grasp is released. Not the muscles which move the elbow, for in this joint flexion and extension alternate with each other during the act of sawing. Not the muscles which pull the humerus backwards and forwards (among which are the two halves of the deltoid), because when the back-pulling muscles are contracted the forward-pulling muscles must be relaxed, and *vice versâ*. Clearly the muscles which are most likely to suffer from prolonged strain in this act are those which maintain the head of the humerus securely in the glenoid cavity, for if the head of the humerus were not firmly

held during the powerful swaying to and fro of the bone, the act would certainly become unsteady. For sawing, also, the humerus must be held slightly removed from the trunk. Now, the maintenance of the head of the bone in the glenoid cavity, and the slight separation of the humerus from the trunk, which are essential for the act of sawing, both demand the contraction of the supra-spinatus.

Duchenne (*Physiologie des Mouvements*, p. 73 *et seq.*), when speaking of the actions of the supra-spinatus muscles, says that it (1), moves the humerus away from the trunk and slightly forward, and at the same time rotates it inwards very slightly, and (2), it depresses the external angle of the scapula. It usually co-operates with the deltoid and serratus magnus in raising the arm, and in the absence of the former muscle the supra-spinatus and serratus magnus combined can raise the arm very considerably. He further says, "The supra-spinatus does not merely help the deltoid in its action by its slight power of raising the arm, but its assistance is needed for maintaining the head of the humerus in the glenoid cavity during the raising of the arm. I have noticed, indeed, in my experiments, that when the deltoid contracts by itself, the head of the humerus has a tendency to escape from the glenoid cavity and to be dislocated downwards." When summing up the actions of the supra-spinatus, Duchenne speaks of it as an "active ligament of the scapulo-humeral joint." It is easy to understand how necessary the supra-spinatus must be for the act of sawing, not only in holding the head of the humerus firmly in the glenoid cavity, but also in holding the humerus slightly removed from the side.

It may be noted that the clavicular portions of the pectoralis major, which was wasted in this case, and amongst the fibres of which there was a tender spot, is also indispensable for the act of sawing, not only by helping in the to-and-fro movement of the humerus, but also in holding the head of the humerus in the glenoid cavity.

In this case of "sawyer's cramp," then, there were found two muscles wasted, both of which were indispensable for sawing, and both of which were presumably subjected to *prolonged contraction* during the act.

What was the nature of the pathological changes? and what was their order in time of occurrence? Were the nerves affected before the muscles, or *vice versâ?* Was the nervo-muscular condition merely the expression of central (spinal cord) change? These questions remain to be answered. The peripheral changes were certain.

Clinical Cases.

A CASE OF CEREBRAL TUMOUR.

BY ALEXANDER BRUCE, M.B.

THE following case is interesting from the absence of the usual symptoms of cerebral tumour, and from the simulation of hysteria, which deceived every one who saw the patient during the last four months of life, even when careful search for gross intracranial disease was made.

L. M., 45 years of age, unmarried, right-handed, formerly a nurse, was sent to me on the 21st of September, 1882, by Mr. Miller, with a history of being nervous and fanciful, and of having had a fit of some sort at each of her last three menstrual periods, and of having in July had an hysterical attack.

Previous History.—Until two years ago she had been remarkably healthy, had been of a cheerful and kindly disposition, and had always had good situations as nurse. So far as could be learned, there had been no previous predisposition to nervous disease, either in patient or in her family. Three years previously she had been present at an accident whereby several men lost their lives, and though she retained sufficient presence of mind to render valuable assistance, she seemed to have been very much affected by the circumstance, as she constantly spoke of it at home. For some time afterwards she was nurse to a very restless child, and, according to her sister's account, was very much overworked. In January, 1881, when it was proposed that she should go to the continent with the family in which she was, a medical man in the country, for a reason I did not learn till post-mortem, dissuaded her from doing so, and advised her to return home, which she did. She then came under Mr. Miller's care, and was found by him to be very nervous and excitable from no apparent cause, till she showed him a tumour in the mamma, which she had supposed was malignant. This,—a small, round circumscribed nodule, without glandular involvement—was removed in

June, 1881. The wound healed perfectly in four days. On microscopic examination the tumour was pronounced by Dr. (now Professor) Hamilton to be non-malignant. For a time after the operation she was very well. In August, her sister informed me that she began to grow very selfish, irritable, and jealous of her sisters; that she would take fits of laughing and crying (especially the latter) for no apparent reason, and that for a time she became so passionate as to be almost unbearable at home. In September, however, she was able to take a situation as a nurse, and for some nine months she seemed in perfect health. She had charge of a very delicate child, that needed constant night attendance; so that for about nine months her sleep was constantly broken. In June she began to get excitable, and to quarrel without cause with her fellow-servants; complained constantly of pains in the back, abdomen, and about one ovary, for which no local cause could be detected, and which were at the time considered hysterical. Finally her excitement became so great that she alarmed everyone by rushing about the house at night screaming, and it was only by firm moral treatment that she could be induced to exercise self-control. She was seen by two physicians, who were of opinion that her case was one of hysteria, and recommended her removal to her own home. During one of her excited turns the first "fit" occurred, and she was said to have rung the bell to summon a fellow-servant to witness it. With rest at home the excitement passed greatly away. At the next period, in August, the second fit occurred; at the third, which occurred a week before time and was excessive, a third "fit." Since this very slight ones had occurred three or four times on some days and sometimes at night. She always knocked her sister up to see those at night, and those in the daytime were stated never to occur in the presence of certain friends, and always before certain others. One occurred during my first examination of the case, and was said by patient and her sister to be quite similar to the others. She felt its onset, said, "There's one coming," grasped my hand with her right hand, shut her eyes, threw her head back, arched her back a little. There was a slight trembling of the body, but no vasomotor change nor frothing at the mouth. She answered a question I put to her, showing (what she afterwards stated positively) that there was no loss of consciousness. The whole lasted for about thirty seconds, and was followed by no headache, or any symptom except slight numbness, which passed off immediately. Our examination went on as before. The "fit" did not seem epileptiform, but such as one sees frequently in hysterical subjects.

Though the history and behaviour strongly suggested

hysteria, Dr. Bennett's case warned me to search carefully for evidence of intracranial lesion. There was found absolutely no alteration of sensibility of general or special sense (smell and taste were unfortunately not examined). Vision and accommodation, good in both eyes. No hemiopia. Pupils equal, active, and of medium size. No ptosis; no strabismus. Ophthalmoscopically fundus healthy; no sign of past or present neuritis. All the reflex functions—tendon, skin, and organic—were normal. There was no headache, and patient said never had been. (Post-mortem, I learned that she had in June complained of throbbing in the head.) No scalp percussion tenderness. Intellect clear, articulation unimpaired; could converse apparently without effort. Memory good. Her manner was excitable, and facial expression strongly suggestive of the hysterical temperament. Tongue slightly furred, slight constipation. Patient stated that she had never vomited. Otherwise I detected no abnormality. There was no sign of, and no reason to suspect, syphilis.

In short, patient seemed simply excitable, and to have fits which might fairly, except for their occurrence at night, be called hysterical. There was complete absence of all the ordinary signs of gross intracranial disease. After watching the case for a month and treating with bromide of potassium and morally, and finding by the middle of October that the fits had almost entirely gone (still happening, in presence of the friends aforesaid), that she had taken regular daily exercise, was sleeping well, and eating well; and that the only symptom she showed was increasing jealousy of her younger sister (who would not allow her to keep house); and after finding on a second examination the same complete absence of symptoms, I felt justified in excluding gross cerebral disease. In the hope of enabling or inducing her to control her temper, and as she was proving utterly unmanageable at home, she was recommended to go to the country for a time. She left town in the beginning of November; wrote after ten days that she was very well, etc. (The letters, which I saw post-mortem, were clearly written and well expressed; and I also learned post-mortem, from the lady in whose house she was, that nothing particular was seen wrong with her). On Nov. 17, she was brought into town; she had had a "fit" (the only one she had had); was said to have "paralysis"; a medical man had seen her, and had recommended treatment by "firmness with kindness." I learned post-mortem that she had summoned a servant to see the fit, and who, when it was over, said, "If that's all your fit, you needn't have troubled me." On Nov. 18 she was quite well in the morning; knitted some reins to send out to the children, without showing any sign of

awkwardness in the use of her hands; went out shopping for four hours. In the evening she began to "talk nonsense," became unintelligible, and was supposed to be going insane.

November 19th.—I saw her in the evening. She was in bed. Mouth twisted slightly to the *left* side, *left* occiputo-frontalis weaker than *right*, slight convergent strabismus, slight weakness of right hand. Legs equally strong. No ptosis: pupils equal, active, of medium size. No optic neuritis. No alteration of sensibility, general or special. Speech peculiarly altered. No ataxic aphasia. Some, especially leading, questions were answered at once, and correctly; others after a pause, and apparently after trying to read in my face what she ought to answer. When she failed in this, she answered as if shamming stupidity. Occasionally she put her hand to her head, and said, "Oh, dear me!" as if annoyed at her loss of memory.

After my visit she got up, walked about for an hour, arranged the bed herself, using her right hand without sign of weakness.

Next day Dr. Brakenridge saw her with me. We found no strabismus, *no twist of the mouth, no inequality in the two occipito-frontales*, or in the *strength of the two hands or legs*. The grasp of the hands was fairly strong. The tendon and skin reflexes were perhaps slightly exaggerated, but equal on both sides. So far as we could judge, sensibility to touch, pain, temperature, on either side was unaltered. Watch heard with right ear at one yard, with left at one yard and a half. The speech had still the same peculiar character, not easily described, but which suggested to each of us that she could answer when she liked, and when she didn't know what was the proper answer to give she "hedged," or shammed to be stupid; thus she answered leading questions at once, and when asked on which leg she felt the prick of a pin best, she said, "It's not so well on that leg, as it's worse on the other." Unfortunately we do not remember any of her other remarks, but they were somewhat similar. She seemed in all cases to understand our questions.

Her whole behaviour, the anomalous nature of the facial pareses, their disappearance, and that of the weakness of the right hand, and the peculiar speech, suggesting irresistibly that she was shamming, and the absence of all signs of gross intracranial disease, seemed to point to a functional cause. A somewhat guarded prognosis was given, and her removal to the Royal Infirmary was recommended.

On the following day she seemed quite well, talked quite rationally during my visit, said there was nothing wrong with her, that she could be treated at home quite well, and finally

began to cry, or rather seemed as if pretending to cry, as if she thought it necessary to make a strong objection to leave home. Her sister told me that after my two previous visits she talked quite well for a time, and then relapsed again; that on the previous afternoon she had had a struggle with her, and that my patient had nearly overmastered her, and that she didn't believe in her weakness.

I saw the patient only once after this—three days before death. She was sitting by her bed-side, with a stolid expression; otherwise she looked well. On my asking, "How are you to-day?" she answered with a rude stare, "Have you been sleeping?" and similarly to one or two other questions. I could not say whether she understood the questions, or whether she took this means of preventing any conversation. Some time afterward she looked as if she had discovered that she had made some mistake (but this may be an error on my part). She could walk quite well.

Dr. Mackie Whyte, resident physician, communicated the following facts. For the first few days she seemed quite well, only hysterical, wouldn't do what she was told, could only be induced to do light work, such as dusting the tables, and when the nurse was by her side, and did so then in a careless, indifferent manner, desisting the moment her back was turned. One day she vomited, and spat into her soup, which she didn't like; was supposed to have done this in order not to have to take it. When scolded for this, did not repeat the offence, except once, on the Saturday before death, when she spat into her tea. On admission: talked quite correctly to Dr. Whyte and the staff nurse: would not do so to a probationer; after a few days, began to be confused at the end of a sentence, would transpose words in a sentence, saying, "Fine—the day," for "It is a fine day." Two days before death she frequently used wrong words—e.g. "jujube" for "hospital" and "a pair of legs" for "two pills." On the day before death her father did not observe anything remarkable in her speech. Once or twice she answered Dr. Whyte as she had replied to me, but on being checked for it gave correct replies. When asked to write what she wished, her writing was unintelligible. She seems generally to have understood what was said to her, and carried out complicated directions correctly. Two days before death, after being warned by the other patients that the nurse would be angry because she spat into her tea and poured it on the floor, she went for a cloth and wiped it up. The day before death, at the morning visit, when told by Dr. Whyte to arrange her hair, which she had left hanging about her shoulders, she did so at once. Two days before death she dragged sometimes one and sometimes the other foot slightly, and vomited

once (next day, the 3rd of December) quite suddenly, when asked to walk round a table. During the night she did not sleep, but got out of bed often; folded and unfolded her clothes.

December 4th.—On getting up in the morning, she slipped to the ground, became unconscious, and, on being put to bed, remained as if sleeping quietly for four hours. Half-an-hour before death, breathing became stertorous, and the pulse could not be counted. There were no convulsions.

Autopsy.—Extract from Dr. Byrom Bramwell's report: "Skull cap natural, dura somewhat adherent to skull at vertex. Convolutions markedly flattened, sulci effaced. Surface of brain sticky, but no lymph. Left hemisphere distinctly larger than the right, the enlargement being in great part due to the swelling of the left temporo-sphenoidal lobe. Weight of brain, 47 ounces. Optic discs natural. On microscopic examination, optic discs perfectly healthy." The brain was handed over to me, hardened for three weeks in saturated solution of zinc chloride, and vertical transverse sections were made at the level of the lines in Fig. 4. A tumour was found extending from the lip of the left temporo-sphenoidal lobe to line 4 (which has just missed it), at the junction of the anterior with the middle third of the pons. The dark line in Figs. 1, 2, and 3 indicates the outline of the tumour, but gives an idea of too sharp circumscription. A naked-eye examination seemed to show that it had grown from the darker part, and to have infiltrated round about, the whitish lines representing probably the remains of nerve tissue. In Fig. 2, below the tumour, there is a hæmorrhage of about the size of a hazle-nut. The surroundings were pushed aside, the basal ganglia with the internal capsule being driven inward, the insula upwards, and the convolutions of the temporo-sphenoidal lobe outwards. The claustum, except perhaps in Fig. 1, was indistinguishable (partly, probably, owing to the method of hardening). The cortex was uninvolved, there being always a depth, after hardening, of $\frac{1}{8}$ to $\frac{1}{4}$ inch between the tumour and the surface. The condition of the first temporo-sphenoidal convolution is most interesting, in connection with the interpretation of the speech peculiarity. In Fig. 1 the white matter is slightly infiltrated; in Fig. 2 rather more extensively, though not so much as in the diagram. In Fig. 3 it had completely escaped, and there was a deep fissure between the tumour and the centrum ovale there. Coronal sections at the level of the tip of the lobe showed that a small part of the tumour had diverged and spread forwards into the island, about half an inch below the surface. Throughout it was impossible to distinguish the growth from the external segment of the lenticular

To illustrate Dr Bruce's Case

Fig 1. Section at level of line 1

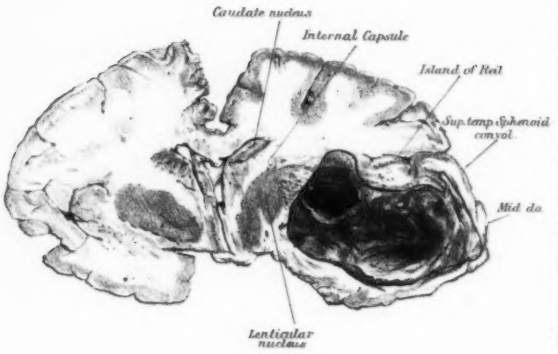


Fig. 2. Section at level of line 2

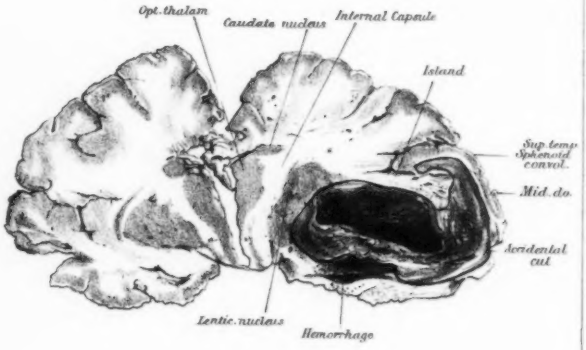


Fig. 3. SECTION AT LEVEL OF LINE 3

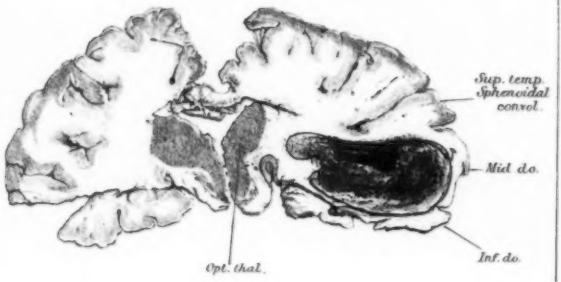
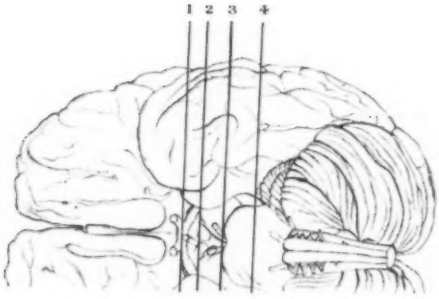


Fig 4. TO SHOW LINES OF SECTION



W & A K Johnston. Edinburgh.

All Hostess for for
Lollipops Shake for
all the Stroppe ride to
Stroppe

I hope for are see
smiles see the Merry
Dresses Shake the children

We are all dressed
Shake the Lollipops
we all ride all Lolly
at three Dressed

nucleus. Judging from a macroscopic examination (owing to the mode of hardening—the only one possible), the starting-point may have been between lines 2 and 3, and the growth from there forwards, backwards, and downwards. Pons, medulla, and cerebellum were much altered in shape by external pressure.

The following facts were learned post-mortem. Dr. Gordon of East Linton, whom she consulted in January, 1881, then found right facial paralysis and anæsthesia, some excitability of manner, and confusion as to names; also deafness in right ear (supposed to be catarrhal), complaint of a confined feeling in the head, sometimes amounting to actual pain, and a history of having on one occasion lost her way near home in a well-known street, and of having to be led home by a policeman. He supposed then that there was serious intracranial disease, but could form no idea of its nature or seat, and he was much astonished to hear that she had recovered sufficiently to again act as a nurse.

Mrs. B.—her mistress during 1881–82—found her a very good nurse, devoted to the child; but easily tired, and then occasionally dragging one or other foot. Sometimes complained of throbbing in the head; finally became excited towards night, and alarmed Mrs. B., who had been recently confined, by shouting through the house at night, and endeavouring to get into her bedroom, which had to be kept locked. During the last month she had frequently complained of nausea, and on several occasions vomited. She was supposed to be very hysterical, and finally to be going insane.

The letter, part of which was lithographed, was written on the day in which she returned from the country.

CASE OF EPILEPTIFORM MIGRAINE.

BY T. CLIFFORD ALLBUTT, M.D., F.R.S.,

Senior Physician to the Leeds Infirmary, &c.

C—— K——, æt. 24, a timekeeper, admitted May 23rd, 1882. His mother, before her death at the age of 62, and one sister who is still living, were subject to occasional attacks of dizziness; and on one occasion he can remember that a brother, without any assignable cause, momentarily lost control over his speech. With these exceptions, there is no family history of nervous affection in any shape or form: no idiocy; no epileptic or other kind of fit; no chorea.

When about eleven months old had three fits in one day, brought on, it is said, by exposure to the sun; but beyond the ordinary diseases of infancy and childhood, he has had no severe illness of any description.

There is no history of syphilitic taint, either congenital or acquired; he has never had any serious blow on the head nor fall; is quite temperate and regular in his habits.

For the last two or three years has had a good deal of mental excitement and worry.

At the age of 18 he went to sea for a year, and on his return went into the building trade, though not as a labourer; for the last eighteen months has been at his present occupation in various factories, the work requiring a good deal of physical exercise and mental activity. He distinctly remembers that when quite a boy he was subject to attacks of dizziness, which have continued up to the present time, and the attendant phenomena to be described below have either developed, or, as is more probable, have been more carefully observed. At any rate, he himself associates the dizziness with his present condition.

The attacks have become more regular and more intense during the last two or three years, and may be said to have culminated in one which occurred in the streets of Selby, about seven or eight months ago, and in which all the subjective symptoms, to be presently described, appeared.

Since this attack in Selby he fancies that his memory has

failed him to a considerable extent, so much so as to oblige him to give up his work.

The seizures are described by the patient in the following manner:

"First there is a semiflexion of the fingers of both hands, and numbness of the feet, which semiflexion and numbness are relieved by the local application of cold, and increased by heat.

"Then the tongue feels numbed and too large for his mouth, and coincident with this tongue trouble is a loss of power of speech; he feels that he knows exactly what he wants to say, but on attempting to say it, probably finds that he is using altogether wrong words, though his articulation of the words themselves is perfect. In the same way with writing; he takes up the pen with the confidence of being able to use it, but finds instead that he has made a series of meaningless scratches on the paper.

"Next his sight gets dim; he can perceive light, but all objects are blurred or only half seen, and the letters of a book seem misplaced. The pain in the tender spot in the left temple, which has continued throughout, and which frequently precedes the attack by twelve or sixteen hours, now becomes much worse, and is often associated with a pain, though slighter, in a corresponding point on the right side.

During the whole of the attack he feels sick and tries to vomit; for he finds that if in the course of a couple of hours or so he can succeed, that there is a very rapid abatement of the symptoms; deep sleep intervenes, and he wakes up in the course of an hour or two, somewhat languid and tired, but otherwise quite well; better, in fact, than he was during the few hours that immediately preceded the seizure.

"If, however, he does not vomit, the symptoms become aggravated; the hands clench tightly; the toes curl downward; dyspnœa, and a feeling of faintness and oppression about the chest, come on, and it is then some eight or ten hours before the attack wears off, the subsidence of the symptoms being associated with drowsiness and deep sleep, as in the former case.

"Consciousness becomes indistinct; it is never completely abolished, and he is clear enough in his own thoughts, though he does not quite realise what is said about him. He retains, even in the most severe attacks, sufficient command to prevent himself from falling, though the spasm of his toes and legs often obliges him to sit or lie down."

When a boy, the intervals between the attacks of dizziness varied considerably; sometimes he would have as many as six or seven in one day, at others only one or two in a week or more.

During the time he was at boarding-schoool (between the age of 10 and 14 years) he does not remember having a single attack, though they invariably recurred on going home for the holidays.

While at sea, and for eighteen months after his return, there was almost a complete immunity from the seizures; and as a general rule he has noticed that they are more frequent and more severe when he is out of work, than when he is busily employed. For the last seven months, up to within three weeks before admission, the attacks have been weekly.

He is particularly liable to a seizure on the day after partaking of a heavy meal, especially if the meal contains anything "rich."

He is a healthy, well-nourished looking man. He describes himself as being very nervous and short-tempered, though during his stay in the hospital he was particularly cheerful and obliging. He seems to prefer to lie upon his right side in bed, though for no definite reason. He suffers slightly from palpitation, and says that on rising from the stooping position he has to move slowly, on account of a sharp pain, which otherwise catches him in the left side immediately below the ribs. Tongue moist, covered with thin white fur. No abnormal physical signs nor evidence of organic disease.

Tactile sensation over the body and limbs appears to be perfect, but over the forehead there seems to be greater sensitiveness on the left side than on the right.

At a point about 1 inch above the external auditory meatus of the left side, and about $\frac{1}{4}$ inch anteriorly, is a very tender spot, the size of half-a-crown, the pain and tenderness of which is much increased during the attacks, though externally there is nothing to indicate any difference between this spot and the surrounding skin. A similar point, the size of a pea, can be made out on the corresponding right side, $2\frac{3}{8}$ inches from external auditory meatus, 3 inches from outer corner of orbit, though this is neither so tender, nor so subject to pain as the spot on the left.

Cannot now drink alcohol in any form without undue excitement, though this was not always the case.

Remarks.—The case here recorded lies upon the confines both of migraine and epilepsy, and illustrates that relation of migraine to other nerve-storms which has been so clearly shown by many modern physicians, and by Dr. Edward Liveing in particular. The attributions by K—— of many of his attacks to "rich food," on the one hand, and to "worry and overwork" on the other, is notable, as is also the banishment of the malady during the months of sea life. The very interesting connections of the symptoms with Dr. Ferrier's centres is

sufficiently clear to any who will read the case with a map of such centres before him, nor will the reader forget how very commonly migraine is associated with aphasia.

A man of some 35 years of age consulted Dr. Clifford Allbutt a few years ago for migraine, the left-sided headache, when more intense, being associated with almost complete aphasia, and with transient enfeeblement of the right hand and fore-arm; and in many cases of migraine a degree of aphasia may be observed. At the same time it must be remembered that, localization apart, general brain fatigue would probably make itself felt in the order of its higher endowments and more complex work.

Speech, being the highest psychomotor function, would thus first feel the failure of general brain-power, and dexterity would be the next in order to suffer. For instance, an English Alpine climber towards the end of an arduous excursion will find the language he speaks worst to vanish from him. If this be German, he may still retain some conversational power in French; but finally this will soon also leave him, and he will be reduced to his mother-tongue. Even this fled from Professor Tyndall on one occasion when, after a grievous climb, he found on entering the Grimsel that he was incapable of articulate speech. Still, when due weight has been given to increasing exhaustion, it seems probable—from the convulsive movements in C. K.'s case, from the visual phenomena of many cases and other special symptoms observed in not a few, that migraine has some partial seat or passage in the brain, and is not altogether a superficial neuralgia.

Finally, however strong the evidence that migraine is a primary neurosis, and however often its recurrences may seem to be due to causes acting directly upon the nervous system, yet it is impossible to ignore the testimony of many sufferers who attribute its recurrence at other times to "bilious" dyspepsia. Moreover, the appearance of migraine in gouty families must not be forgotten, nor the curious fact that the migraines of gouty families are often, to all appearance, the most purely neurotic in character.

brought up. It was not preceded by epigastric pain. The last attack occurred two years ago.

Pains between the shoulders, like a knife or pins running into him, began three or four years ago: he has had pains in the legs for two years. He says he has a "nervousness" in his legs, and sometimes explains this to mean that a sudden pain catches him while walking, and makes him unsteady.

In November 1881, while walking, the nervousness caught him and he fell; he got up and walked home (a quarter of a mile), and even went out again; but he again fell, on account of the pain, was carried home and went to bed. He could move the leg in bed, though not so well as now. It began to swell (he thinks in two or three days' time) first in the upper and outer part of the thigh, then in the groin. There was (he says) no redness or tenderness, nor any pain except the "nervousness." It appears that there was some inco-ordination before the hip became affected; for he states that for two or three months previously he would hesitate on coming to a crossing, and felt that he could not walk in the dark.

Present condition.—He is gray and thin; looking older than 58. Very deaf; somewhat apathetic in his ways. Pulse 90; arteries hard and tortuous: a systolic murmur at the heart's apex, and an accentuated second sound over the aorta. No albuminaria. He totters when his eyes are shut, or when his feet are put together. Vision of right eye good (excepting slight hypermetropia): optic disc and fundus normal. There is an opacity of the left cornea. Pupils 2 mm. in diameter, no contraction to light, and very little during accommodation. No squint. After removal of a plug of wax from either ear, the hearing distance was, for right ear—contact; for left, 8 inches (watch of 4 feet or more): tuning-fork on forehead or nose best heard with right ear. Both tympanic membranes are opaque and thickened; the right so much that the outline of the malleus cannot be traced. There is some numbness of the feet, but no actual impairment of sensation, either of touch or pain. The cutaneous reflexes are present (the right plantar better marked than the left): the patellar tendon reflexes are absent on both sides.

In Scarpa's triangle (left) there is a swelling, roughly oval, somewhat doughy, over which passes the femoral artery. The great trochanter is thickened, and lies nearly an inch above Nélaton's line. The thigh is movable upon the pelvis in every direction, and a grating can be felt as it is rotated outwards. There is no pain. The position of the limb (as the patient lies with his legs extended) is very much that of the other limb; and can be varied by him: there is over an inch of shortening. These conditions appear to indicate that there

TWO CASES OF TABES DORSALIS WITH JOINT-DISEASE.

BY J. A. ORMEROD, M.D. (OXON), M.R.C.P.

THE subjoined cases illustrate the two varieties of this disease, which Charcot describes as "malignant" and "benign." In the first, the disease advanced to destruction of the joint, producing a kind of dislocation; in the second, the joint recovered so far as to be practically, though not perhaps anatomically, sound, and that too while the other symptoms of the disease were progressing.

CASE I.—Vomiting; pains between shoulders and legs; unsteadiness of gait; sudden fall, followed by swelling of thigh; disease of hip-joint.

Wm. B., 58, ship-joiner, came to the out-patient room at Queen's Square on Aug. 16, 1882; his chief complaint being lameness of the left leg; he walked with support, the left leg seeming to give under him. He said that nine months previously, while walking, a sudden pain caught him in the left hip-joint, and caused him to fall; the thigh swelled up, and he had been lame ever since. Examination showed that there was some affection of the left hip-joint. From the small and inactive character of the pupils, and the absence of the patellar-tendon reflex, it was surmised that the case was one of locomotor ataxy, and he was subsequently admitted into the hospital, where, by the kind permission of Dr. Buzzard, he remained under my care.

The account taken shortly after admission (October), for much of which I am indebted to Dr. Coxwell, is as follows:

History.—He considers that his health was pretty good till two years ago. Ten years ago he had gout in the left foot. He has had gonorrhœa and a bubo, but not syphilis, to his knowledge.

Five years ago he began to have attacks of vomiting; these used to last a day or two, or sometimes a week, with intervals, roughly speaking, of a month. The vomiting might occur before, after, or independently of food; watery stuff was

noticed a large painless swelling there. The joint is now full of fluid, bulging on either side of the patella, and for some inches above it. The swelling does not extend into the muscles of the thigh or leg; the outline of the distended synovial membrane is clearly mapped out. There is no redness, heat or tenderness. There is no crepitation or grating felt on manipulation, but he says himself that it "cracks." The account he gives of it is this: a week before the swelling came on he got out of bed in the night, and jammed his knee against the bedstead: he had some difficulty in getting it free. A few days after, while walking in the street, the knee "slipped." Nothing further followed till he found the knee swollen in the morning. There was no pain.

The subsequent course of this joint-affection has been as follows:—During the next fortnight the swelling went down a little, becoming less tense: on November 1st, slight swelling was apparent above the limit of the synovial membrane. During December the knee became more swollen than ever, measuring 15 inches as against 12¾ on the left side. During January, February, and March, 1882, the knee-joint fluctuated in size, subsiding on the whole; but there was well-marked effusion above it, beneath the muscles of the thigh. By July 18th, the effusion both in the knee-joint and in the thigh had subsided, and the condition now (November 1882), which promises to be permanent, is this: largest girth of right knee, 13 inches; of left, 12½: in the right knee some thickening both of the bones and of the synovial membrane, but movements natural; a shower of crepitation to be felt when he flexes and extends it. No pain. The muscles of the left calf and thigh look smaller than those of the right; on both sides, however, they act to the faradic current. There is a patch of superficial ulceration at the inner and lower part of the left thigh, produced, he says, by striking the other thigh against it. I may add that in the left great toe-joint (previously alluded to) there is more lateral mobility than in the right, and the nail is thick and horny.

In other respects, the nervous disease has progressed since the patient first came under observation. Thus in November 1881, a "mistiness" of the left (previously sound) eye was complained of, and the vision has since failed gradually. Now there is well-marked optic atrophy; colour of disc, grey-yellow; edges well-defined; arteries perhaps rather small; vision almost nil. The vomiting and diarrhœa seem to have stopped permanently.

The inco-ordination of the legs has become extreme, and is spreading to his arms. In November 1881, shooting-pains in the arms were complained of; in March 1882, numbness of the

has been some destruction of tissue at the head of the femur, producing displacement upwards, if not actual dislocation, of the shaft.

The muscles of the left thigh look smaller than the right (girth at corresponding points R. = 13 inches: L. = $12\frac{1}{4}$): but the thigh-muscles on both sides contract under the faradic current. There is some thickening about the metatarso-phalangeal joint of the right great toe: the gout, according to the patient, was in the other foot.

Since admission there has been slight subsidence of the tumour in the groin, and he has become rather firmer on his legs: he has had little pain or trouble of any kind.

Case II.—Girdle pain: affection of toe: paræsthesiæ and inco-ordination of legs: vomiting, diarrhœa: affection of knee-joint: optic atrophy: numbness and inco-ordination of hands.

[Notes of the earlier condition of this patient were published by me in the Reports of St. Bartholomew's Hospital, vol. xvii. p. 42. His condition has materially altered since that time.]

Henry S., 47, mason; out-patient at Queen's Square Hospital. When first seen (August 3, 1881) there was well-marked inco-ordination of the legs, anæsthesia of the greater part of the feet: absence of patellar tendon reflexes: there was a cataract in the right eye; the pupils were small, unequal, contracting neither to light, nor during accommodation.

He had had tightness round the abdomen for eighteen months; shooting-pains (right leg and groin) only a few months.

He had been subject for four or five months to attacks of vomiting, lasting two or three days, recurring about once a fortnight. This vomiting recurred twice while under treatment (August and October, 1881), being accompanied on the first occasion by diarrhœa after food, and throbbing sensation at the rectum, with some kind of discharge.

There was said to be no joint-disease: but it was noticed that in the left great toe the bone of the first phalanx and the distal extremity of the metatarsal bone were thickened. He said that (in 1880?) he one day took off his boot, and found the whole of the toe swollen and black; the nail came off in a week or so. There was no pain, and nothing to account for it. He has never had gout, but had what he calls slight rheumatism of that foot some years ago.

There was no direct history of syphilis: he had one child living, about eight years old, three born dead since that time.

On October 19, 1881, he came complaining of his right knee-joint. Ten days previously, on rising in the morning, he

fingers; now, when he puts food to his mouth, it often goes to his nose.

His wife says he has had a kind of fit, in which he seemed to lose consciousness and laughed. This once happened before; and he used formerly to talk strangely.

As a resemblance (perhaps superficial) to the other case, I may mention that he is slightly deaf with the right ear. The membrana tympani is thickened and irregular, showing a small perforation in its centre. He had formerly discharge from this ear, and has complained throughout of noises in the head.

This affection of the joints appears to be distinguished by the clinical, rather than by the anatomical, characteristics, and notably by the manner of its onset. The points laid down by Charcot in his lectures are—that it occurs at an early stage of the nervous disease, before inco-ordination has set in; that it arises without apparent cause, such as a blow or a fall, and without premonitory symptoms, except perhaps crackling; that although the onset is acute, it is unaccompanied by pain or sign of inflammation; that over and above the swelling of the joint, there is swelling of the limb, partly due to effusion, partly to solid induration.

The two important features—painlessness and œdema of the limb—are sufficient, I think, to characterise the joint-affection, in the cases just detailed, as different from an ordinary acute synovitis. As regards the stage of the spinal affection, well-marked ataxy was observed in Case II. before the knee was attacked (the toe may possibly have been affected before ataxy set in); and in Case I. the history makes it probable that the patient was ataxic before the hip-disease began. A possible connection with traumatism cannot be eliminated in either case; in Case II. either a blow or a slip; in Case I. a fall, probably occasioned by the sudden giving of the legs, which so often occurs in ataxic patients. Debove has a theory[1] that in these cases the head of the bone is diseased, so that slight strains are sufficient to tear off minute pieces of bone from the point of insertion of the capsule, in this way causing synovitis.

Lastly, the coincidence of joint-disease and of gastric crises, upon which Dr. Buzzard has insisted,[2] finds an illustration in these two cases.

[1] 'Archives de Neurologie,' July 1881.

[2] 'Clinical Lectures on Diseases of the Nervous System,' pp. 251 foll.

TWO CASES OF MÉNIÈRE'S DISEASE.

BY ALEXANDER M. M'ALDOWIE, M.D.

CASE I.—On the 10th of December, 1880, I was sent for hurriedly to see Mrs. H—, aged 43, who had fallen down a flight of stairs. I found the patient in bed vomiting severely. She complained of giddiness, nausea, and a loud singing noise in the left ear. There were several bruises on the arms and side but none on the head. The pupils were natural and reacted readily to the stimulus of light; the intellect was clear. Any attempt to raise the head brought on a severe attack of vertigo, the patient experiencing a sensation as if she were falling backwards. Each of these attacks ended in a fit of vomiting. On examination a watch could only be heard one inch from the left ear, the hearing in the right ear being normal. There was a slight purulent discharge from the left auditory meatus.

The patient stated that she had suffered from deafness and noise in the ear for more than six months. She describes the noise as being of a singing or buzzing character. She was subject to fits of giddiness accompanied with nausea and vomiting. Just before and during those exacerbations of her complaint the noise became very much increased. She had never fallen before during a fit. She stated that she did not trip her foot at the top of the stairs but fell down from giddiness.

I ordered a saline purgative, and a blister to be applied to the back of the ear.

The following day the noise remained as before, although the giddiness and nausea were less severe. There had been no vomiting since two o'clock A.M.

On the third day the patient returned to her home in Liverpool.

CASE II.—Sarah H—, aged 31, married, came to me in August last complaining of attacks of giddiness and sickness. About two years before the patient had a severe fall, injuring her right side, but she stated that her head did not sustain any

injury. She suffered a good deal from shock after the fall. Ever since, she had been troubled with vertigo and a constant ringing noise in the right ear. She suffered much, at irregular intervals, from attacks of "swimming in the head," when her body seemed to rotate round a vertical axis. She could not state whether the rotation took place from right to left or vice versa. Each crisis was heralded by a shrill whistling noise in the right ear. These seizures almost invariably terminated in a fit of vomiting.

She could hear a watch only when in contact with the right ear, the membrana tympani was thickened and opaque. The left ear was normal. The heart was dilated, the first sound prolonged and harsh; the pulse 124, weak. Tongue clean, appetite good, bowels regular.

I ordered iodide of potassium to be taken internally, with counter-irritation over the right mastoid region. The patient gradually improved, the "fits" became milder and less frequent, and in about six weeks she left off treatment, being nearly well.

Remarks.—The above cases of Ménière's disease are interesting, specially from a clinical point of view. They are not examples of the grave form of this malady, described by some French authors, yet the symptoms of disease of the semi-circular canals are well marked in both cases. This disease in its less severe form is apt to be overlooked in general practice and the case treated as one of gastric vertigo or epilepsy. Case II. had consulted several medical men in this district and had invariably been informed that she was merely suffering from indigestion. She had undergone several courses of treatment but without deriving any benefit. This case illustrates the value of counter-irritation strongly recommended by Professor Charcot and other writers. Charcot consides quinine almost a specific for this disease. I regret that I had not read his lectures at that time and therefore did not test its value in this case.

In Case I. the patient maintained that the symptoms were the result of injury to the head produced by the fall, and it was only by persistent questioning that I elicited the fact that the hearing was impaired on the left side, and that she was subject to attacks of vertigo.

It is interesting to note that in one case the patient invariably experienced the sensation of falling backwards, whilst the other patient always felt as if her body were rotating on its vertical axis. Professor Crum Brown states that one canal can be affected by and transmit the sensation of rotation about one axis in one direction only, and that each pair is sensitive to any rotation about a line at right angles to its

plane or planes, the one canal being influenced by rotation in the one direction, the other by rotation in the opposite direction. Notwithstanding this the cases narrated above do not appear to indicate disease limited to one canal. For cases are on record where the motor hallucinations changed their character from time to time; the rotation at one period being always apparently forwards, whilst at other times it would either seem to take place in the contrary direction, or round a vertical axis.

with one semi-circular canal (external) and two ampullæ; but while the saccus may be compared to the sacculus of other animals, the single canal may perhaps rather represent the utriculus.

In Petromyzon (fluviatilis) there are two semi-circular canals corresponding to the anterior and posterior of higher animals, with two ampullæ presenting a peculiar trifid subdivision, the middle part of which in each corresponds to the true ampulla and presents the crista acustica. The commissure of the two canals is probably the same as in other animals, but the nature of the vestibule is somewhat doubtful. There is probably an aqueduct of the vestibule or endolymphatic duct, and it may be that the elongated macula acustica combines those of the recessus utriculi, the sacculus and the lagena cochleæ; but as these are not separate some doubts may exist regarding them, and there is thus a considerable gap between the structure of the hearing organ in Cyclostomata and that of other fishes.

Among the remaining fishes, various as is their structure in many other respects, it is surprising what general similarity prevails in the structure of the hearing organ.

Among the Ganoids, Acipenser is the forerunner of the Osseous ganoids (Lepidosteus) on the one hand, and of the Teleoster on the other, but in the structure of the labyrinth it comes very near to the Teleostei. A large canalis utriculo-saccularis and a ductus endolymphaticus are present. The otolites have not yet acquired the hardness which they possess in Teleostei.

In the Osteoganoids there are true hard otolites. The lagena is only slightly separated from the sacculus. The endolymphatic duct is wide and ends as a cæcum superiorly.

The variations in the form of the hearing organ in Teleostei are so great that it is difficult at first to reduce them to one type, but a closer examination shows them to be essentially similar. Hasse has distinguished two types among them, viz. 1, that of the Perch, and 2, that of the Cyprinoids; and there is perhaps a third also, viz. that of the Clupeidæ.

Great variation in the size of the sacculus is to be found: it being very large in Gobius and Trachinus, very small in Lens, and reduced to a minimum in Exocetus. The lagena also is very variable; in Esox and Belone it is scarcely separated from the sacculus, in others as Pagellus, Scomber, Clupea, forming a considerable bursa-like swelling, and again in others, as Gadus, Lophius and Raniceps, flask-like and opening into the sacculus by a narrow tube.

In the Lophobranchii (Siphonostoma, Hippocampus) the sacculus is not separated into an upper and lower part, and this contrasts strongly with the disposition in Anarrhicas in which the two parts are nakedly distinct.

In connection with the canalis communicans or utriculo-saccularis Retzius finds, in confirmation of his previous researches and in opposition to the statement of Hasse, that in 11 of the 33 Teleostei described the communication exists. In Clupea and Corregonus it is just perceptible and in the remaining 20 it is

Reviews and Notices of Books.

Das Gehörorgan der Wirbelthiere—Morphologische histologische Studien. Von GUSTAF RETZIUS, Professor, &c., in Stockholm. Folio, Stockholm. Feb. 1881. 1. *Das Gehörorgan Fische und Amphibien* (with 35 plates).

WE regret having been so long giving a notice of this important work which may be regarded as a masterpiece in its department of Comparative Anatomy. It is dedicated to the memory of the author's illustrious father, Andreas Retzius, and certainly no more appropriate tribute could be offered by his son to the untiring genius for research of that most able and amiable anatomist.

This part forms the first instalment of a complete work on the organ of hearing in vertebrate animals which its author had planned as early as 1871, but which at one time he had almost been inclined to abandon after the publication of several of the admirable memoirs of Hasse on the same subject. But fortunately for science he was led to continue his observations and to bring them together for publication; and it may be confidently hoped that the second part of the work may, according to the author's expectation, ere long appear. The part at present under review comprehends fishes and amphibia, and the remaining part will embrace reptiles, birds, and mammals.

The work presents all the characters of having been executed with great care, both as regards the 220 pages of descriptive text, and the 35 beautiful plates, containing in each on an average between 13 and 14 figures, and in the careful reference letters and description. In fact it may be looked upon as a model for such works both by the clear description of the large amount of original works which it affords, and by the copious reference to the other researches the publication of which has preceded that of the author.

Through the kindness of a number of his friends and correspondents and his own opportunities, the author was enabled to include in his investigation and description as many as 48 fishes and Dipnoi and 15 Amphibia; and he promises the description in the succeeding volume of 22 reptiles, 10 birds, and several mammals.

The following is an abstract of the principal statements made by the author in the first part of his treatise:

In the Cyclostomata the structure of the organ of hearing stands so much below that of other fishes that it scarcely admits of comparison with them. In Myxine there is a saccus communis

The single or undivided macula acustica neglecta lies in the base of the utriculus.

There is no distinct lagena cochleæ, but there is a separate papilla acustica lagenæ on the hinder and inner wall of the sacculus.

The endolymphatic duct was not found, and if it exists it certainly does not reach the surface of the head as in Elasmobranchs.

The utricle and its superior sinus are not divided vertically as in Plagiostomes. The ear cavity is, as in Teleostei, Ganoids and Chimæra, open to the brain cavity. The otolites consist only of small separate crystals. The ear of Dipnoi differs therefore considerably from that of Ganoids, and approaches more nearly to that of Elasmobranchs, especially of Holocephali or Chimæra. It seems as if the Dipnoi together with the Holocephali constituted a common but later divided lateral branch of the "principal phylogenetic line." As regards the structure of their organ of hearing they cannot be placed, as was formerly attempted, in the same group with the Ganoids.

Between fishes and Amphibia there is of course in respect of the organ of hearing a gap not easily filled up; but yet not so great as might previously have been supposed.

In the lowest Urodeles the structure of the organ, as in Proteus, Menobranchus and Amphiuma, recalls forcibly that of Acipenser, although a separated lagena cochleæ exists in the Urodeles and there are other minor differences. They might be considered to have the properties of the Proganoids, which are inadequately represented, and the many intermediate members are unfortunately not known. With the divergent Dipnoi and Elasmobranchs on the other hand they have much less in common.

All the Amphibia, Urodeles as well as Anura, have the ear capsule closed on the inner or cerebral side by a partition wall. The lower Urodeles are especially characterised by the existence of a true perilymphatic space, limited by an external periosteal covering, and connected with the cerebral cavity by a tube (ductus perilymphaticus, Hasse) which passes through a special foramen. This space and its tubular outlet have nearly the same form in the higher Urodeles (Siredon, Salamandra), in which they were discovered by Hasse. The same observer also discovered the space and tubular outlet in the Anura, but in these animals the latter is dilated into a sack, and there also runs a long and somewhat complicated tube into the perilymphatic space where it opens.

In Urodeles and Anura, as in fishes, the ductus endolymphaticus proceeding from the sacculus goes, as shown by Hasse, through the apertura aquæductus vestibuli into the cerebral cavity, there to dilate into a comparatively large folded sack surrounding the brain but closed against the cerebral cavity.

The other morphological components of the membranous part of the organ of hearing in the lower Urodeles are the same as in fishes. There is a division into a superior and an inferior part, and these communicate with one another by means of a hole or tubular aperture, canalis utriculo-sacculus. Upon the projecting

entirely absent. In two thirds of these fishes there was present the macula acustica neglecta described by the author in a previous work, while it is absent in eleven. It is singular that in allied tribes such differences should exist.

The form and structure of the otolites would form an interesting subject if treated systematically; but the author has not been able to go into it farther than in connection with the other parts.

In the Elasmobranchs Retzius points out three very obvious types in the structure of the organ of hearing, viz. that of the Holocephali, of the skates, and of the sharks. The type of the Holocephali (Chimæra) forms an interesting transition to that of the Plagiostomes and through these to that of other fishes.

In all the Elasmobranchs the ductus endolymphaticus forms a pervious tube opening upon the surface of the head. In some it is straight and simple, in others it is narrowed or bent horizontally near its termination. There is also in all a separation of the recessus utriculi from the utriculus and from the outer and anterior ampullae, which is remarkable as compared with the form in Ganoids and Teleostei.

In Chimæra there is only a small opening, and the anterior and outer ampullæ open into the utriculus directly and not into the recessus. In the sharks there is often a pretty long tube between the utricle and the saccule, and there are in other instances interesting gradations in the development of this canal.

The macula acustica neglecta is present in all the examples of this group of fishes examined by Retzius. In Chimæra it constitutes a small nerve termination situated at the hinder end of the canalis utriculo-saccularis; but in skates and sharks the position is different, in consequence of the separation of the utriculus and the superior sinus. In the Elasmobranchs, in contrast with what is observed in the Teleostei and the sturgeon, the macula acustica neglecta is single or undivided.

As regards the lagena, this part is not separated from the sacculus in Chimæra, and its papilla is still in connection with the macula acustica of the sacculus. In the skates and sharks on the other hand the lagena forms a distinct bursa-like swelling of the sacculus, its papilla being in the skates only notched off from the macula acustica, but distinctly separated in the sharks.

In the sharks and skates, as is well known, the cartilaginous ear capsule is separated from the cranial cavity by a wall, but in Chimæra there is no partition so that it is a part of the same cavity with the brain.

In the Dipnoi there are interesting relations which receive their explanation from the study of the Elasmobranchs and particularly of Chimæra. In Ceratodus and Protopterus, and more especially the last, we find a very large recessus utriculi, not communicating directly with the utriculus or with the anterior or outer ampullæ, but united by an oval canal, (can. recessu-saccularis) with the sacculus, and through its intervention by the canalis utriculo-saccularis with the utriculus itself. The anterior and outer ampullæ open as in Elasmobranchs into the anterior end of the utriculus.

median side of this opening there is in the lower Urodeles a small nerve-twig and termination (derived from the ramulus ampullæ posterioris) lying as in Chimæra and to some extent in Plagiostomes on the wall of the posterior canal, and this Retzius regards as certainly corresponding to the macula acustica neglecta of Elasmobranchs and other fishes.

As regards the true cochlear part there is not, with the exception of the lagena and its papilla acustica which are present in all amphibia (except Cæcilia), any further place of nerve termination in the lower Urodeles, as Proteus, Menobranchus and Amphiuma; but in the higher, as Siredon, Menopoma, Pleurodeles, Salamandra and Triton, there is the first appearance of a separate papilla acustica basilaris cochleæ. But Retzius does not agree with Hasse and Kuhn in describing this as a true basilar part of the cochlea with its cartilaginous framework. But in the Anura he states that there is a well developed and distinctly separated pars basilaris cochleæ, which has been well known, since it was described by Deiters and Hasse, with the papilla acustica and cartilaginous rim or border, the thinner wall of which Retzius regards with Hasse as corresponding to the membrana basilaris. He has not succeeded in finding transition forms between Urodeles and Anura, and he does not believe that such exist.

Retzius describes shortly the accessory parts of the organ of hearing in Amphibia, into the details of which it is not necessary to enter here. He calls attention to the peculiar saccularly dilated duct (ductus fenestræ ovalis), which in the frog runs from the perilymphatic space to the cartilaginous plate of the columella and projects in the fenestra ovalis, and to the fact that a similar duct or diverticulum of the perilymphatic space is already more or less developed in the other Anura and the higher Urodeles.

Throughout the whole work Retzius has given minute descriptive details of the histological structure of the terminal nerve apparatus and other parts, all of which are illustrated by clear and appropriate figures drawn by himself from microscopic specimens. Space does not admit of our following the author in this description, and as he means to return to the whole subject of the finer structure of the membranous labyrinth when he has completed his second volume, we shall probably have another opportunity of reverting to it.

Retzius concludes his description with a reference to a "phylogenetic line" or diagram which he has given, and in which the affinities of the several groups and genera are traced from the lowest point upwards and in diverging series. The principal line passes through the Proamphibia, Urodeles, Posturodeles, Proreptilia, and thence on to higher animals, and the successive lateral offsets from this line include the Myxinoids and Petromyzons, the Proganoids, Sturgeons, and Osteoganoids and the Osseous fishes on the one side; the Dipnoi, Proselachii, Holocephali, skates and sharks on the other, followed by Proteus, Siredon, Triton, and Anura.

ALLEN THOMSON.

LES PTOMAÏNES.—*Recherches chimiques, physiologiques et médico-légales.* Par I. GUARESCHI et A. MOSSO, de l'Université de Turin. Première partie. 8vo. pp. 55. Rome, Turin, Florence: H. Loescher.

THE organic alkaloids formed by the decomposition of the nitrogenous constituents of animal bodies, and to which the name of ptomaïnes has been given, have become of great importance from a medico-legal point of view. Our increasing knowledge of vegetable poisons has increased the possibility of new alkaloids which cannot readily be detected, being employed for the purpose of criminal poisoning. As alkaloids are formed, however, by the natural decomposition of the body itself, it would be quite possible that an innocent person might be convicted of poisoning from an alkaloid being found in a dead body, although no such had been given; and on the other hand a guilty person might escape on the plea that the poison actually found was a ptomaïne formed in the body and not administered at all.

In order to ascertain precisely the reactions and physiological actions of ptomaïnes, a committee has been appointed by the Italian Government to investigate the subject. The present work is the first-fruit of its labours, and the excellent way in which the subject has been worked out shows how wisely the committee has been selected. It would be here out of place to enter into the chemical part of the investigation, and we shall mention only those points that are of physiological interest. One of the first of these is the existence of organic alkaloids in different kinds of alcohol. E. Ludwig found trimethylamine in Austrian wine, and Krämer and Pinner found in alcohol a mixture of bases belonging to the picoline series, amongst others collidine. Guareschi and Mosso have also found in ordinary alcohol an organic alkaloid, probably either pyridine or a homologue of it. In amylic alcohol they found pyridine. It seems not impossible then that some of the injurious effects of amylic alcohol may be due to the action of organic bases contained in it.

From putrefied brain they obtained an extract which has an action on the motor nerves analogous to that of curara. It is, however, much less energetic. A much larger quantity of the substance is necessary to produce the same effect, and poisoning even when very complete lasts for a much shorter time than that of curara. Its action upon the heart is to render the pulsations stronger and slower. There is no period of acceleration, and the increase of energy lasts for a considerable time.

From putrefied fibrine they extracted a ptomaïne which produced in frogs complete immobility, muscular relaxation and loss of reflex, even when the muscles and motor nerves still responded regularly to stimulation by a faradic current.

A curious point was the agreeable odour which the poisoned frogs exhaled, resembling that of orange flowers. A finch poisoned by the ptomaïne vomited, trembled and showed diminished sensibility,

but soon recovered. A rat weighing 76 grammes exhibited no symptoms after the injection of 20 milligrammes of the extract of putrefied fibrine. The difference between the activity of this ptomaïne and the ordinary organic alkaloids is very great, so that one may consider it as quite innocent in doses where coniine, nicotine or curara would be promptly lethal. Hydrochlorate of ptomaïne obtained from fibrine paralyzes the motor nerves like curara, but probably acts also on the muscles. When the muscle of a frog poisoned by it is irritated at regular intervals, it does not respond to each irritation by a contraction as an unpoisoned muscle would do. The contractions are sometimes high, sometimes low, and sometimes are absent. The excitability of the muscle becomes rapidly exhausted. This condition corresponds exactly to that described by Harnack in poisoning by lead and some other metals.

The similarity of the action of ptomaïne to curara leads the authors to discuss more particularly the action of curara itself, and they consider that the effect of this poison is not something *sui generis* but resembles the natural death of the organism. They found that the sciatic of an unpoisoned frog remains for a certain length of time at its normal level, and when it is irritated by opening shocks so feeble as to be hardly felt upon the point of the tongue a regular series of contractions is obtained from the muscle. This is the first stage. During this it is always easy to obtain regular contractions if the stimulus be sufficiently strong, but if the stimuli are very feeble, the contractions may neither be equal nor decrease regularly; they may be sometimes higher, sometimes lower, and even be wanting entirely.

In the second period, that of diminished excitability, this condition is still more marked, and in order to get regular contractions the stimulus must be increased in power. This second period may be observed in a frog which has been decapitated several hours previously or in one where the nervous excitability has been diminished by the influence of curara, of ptomaïnes, and of other nerve poisons. Whatever may be the poison employed, the result of poisoning by any drug which lessens the irritability of the nerves is exactly the same as that due to the natural death of the nerve. Under certain conditions Luciani has observed a curious irregularity in the beats of the heart which occur in groups, separated by intervals of quiescence. This takes place under the same conditions and is probably analogous to the irregularity in the contractions of voluntary muscles during the second state of dying, viz. that of decreasing excitability.

In the third period the nerve is no longer excitable by strong opening shocks which are isolated and separated by an interval of about a second, while it is still excitable by shocks which are more feeble but frequently repeated, like those of a Du Bois Reymond's coil.

This period is followed by the last, that of the death of the nerve, which completely loses its excitability, while the muscle still contracts on direct stimulation. Such is the course of the death of the sciatic nerve in the frog.

The action of poisons has nothing specific: they do not change the order of these four periods, they can only make them appear more quickly and accelerate the death of the nerve. The action of curara resembles natural death even in one point which has been supposed to be specific, viz., the continued sensibility of the sensory nerves after the motor nerves have lost their power. This condition is, however, produced, as the authors show, by simple compression of nerve trunks. When the lumbar nerves going to one leg of a frog are compressed for a short time by a small bull-dog forceps, irritation of that leg will produce movement in the rest of the body, while the leg itself remains paralysed and motionless.

Weir Mitchell has noticed that in wounded nerves the motor power may be abolished, while the sensory function is little altered. Erb has often seen in contusions of nerves sensibility re-established, while voluntary motor power was lost for ever. Lussana says that one characteristic symptom of rheumatic neuralgia is paralysis of the muscles and exaggerated sensibility of the corresponding parts.

The authors believe that it is this great vulnerability of motor nerves, and not an anatomical reason which furnishes an explanation of the readiness with which paralysis of the facial and laryngeal muscles occur in man, while disturbances of sensibility are comparatively very rare. The difference between the vitality of motor and sensory nerves becomes clearly evident from the effect of compression of the radial nerve which is one of the most common causes of paralysis of the muscles of the arm and of the hand; the paralysis being often complete while sensibility is hardly altered, if at all.

A study of other poisons supports the author's theory of the action of curara. A number of substances have been shown to have an action like it. Rabuteau showed iodide of tetramethyl ammonium; and Bufalini tetraethyl ammonium to have a similar action. V. Meyer obtained a similar substance from beer, and Bocci obtained another from human urine. In lead poisoning also the voluntary movements of the extensor muscles are abolished while sensibility is preserved.

The action of anæsthetics, which at first seems to be in opposition to this hypothesis, really agrees with it: thus in poisoning by chloral the posterior extremities preserve their sensibility after they are paralyzed, and even after the forelegs or head are paralyzed, changes in respiration show that sensory impulses are still nervously transmitted. The same thing has been noticed by narcotising with chloroform or ether, or by injecting pyridine or alcohol into the abdominal cavity. Even when the respiration has become too feeble to show any reflex change, the sensory nerves may be still showed to preserve their properties, for the pupil dilates at every irritation of the extremities.

The authors are no doubt right to a great extent in their views with regard to curara; and they may be also right to a great extent in considering that motor paths are sooner paralysed than sensory.

But if this be so, we can hardly do otherwise than assume that sensory cells are sometimes rendered functionally inactive before motor, as otherwise it is difficult to understand the relief of pain by morphia or anæsthetics while motor power still remains. It seems probable, however, that some drugs affect sensory sooner than motor fibres, for Pratt having surrounded the sciatic nerve of a frog with a paste of theine and water found that irritation of the left foot caused no reflex action while irritation of the spinal cord caused motion in both feet alike.

The curara-like action of ptomaïnes is a subject of great practical interest. In a paper on indigestion as a cause of nervous depression (Practitioner, vol. 25, pp. 265) the reviewer pointed out the resemblance between the symptoms of poisoning by paralyzing agents, and the muscular weakness and languor which occur in certain forms of indigestion, and drew attention to the fact that these symptoms of poisoning in indigestion might be due to substances absorbed from the intestine. At that time he was inclined to attribute these symptoms to peptones which have been shown to have a poisonous action, but the researches of Guareschi and Mosso appear to show that it may not improbably be due to ptomaïnes which have a curara like action. This seems all the more likely because not only has Bocci, as already mentioned, found a substance in the human urine which paralyzes frogs, but Bouchard, Rev. de Med. 1882, No. 12, has found ptomaïnes both in the urine of patients suffering from infectious diseases and in normal urine. He has also found them in large quantities in the fresh fæces of healthy persons as well as in diarrhœa and typhoid stools. The proportion of ptomaïnes in the urine to that in the fæces is as 1 to 40 or 50. These ptomaïnes he thinks are probably formed in the intestinal canal and absorbed into the blood. These researches afford an explanation of the acknowledged utility of purgation in nervous depression and especially of the utility of calomel which will not only remove ptomaïnes from the intestinal canal by its purgative action but will tend to prevent their formation as Wassilieff (Zeitschr. f. physiol. Chem. VI.) has shown it to possess a very considerable power of arresting fermentation and destroying micro-organisms.

T. Lauder Brunton.

De la Démence Mélancolique (*On Dementia Melancholica. A Contribution to the Study of Periencephalitis, Chronic and Localized, and to the Study of Psychic Cerebral Localization*). —By Dr. A Mairet, Professeur Agrégé of the Montpellier Medical School. 296 pages and xi. planches lithographiées. Masson, 1883, Paris.

The general idea contained in this book is the following:—Is it not possible to ascribe certain psychical symptoms which one may conveniently call *Dementia melancholica*, to functional disturbances

and to diseases of the brain-cortex? M. Mairet, who, it must be said, is *médecin-adjoint* to an insane asylum, says he has often remarked among many insane some psychical disturbances, together with some limited anatomical diseases. These disturbances he ascribes to the diseases.

The disturbances are the following: melancholia of a delirous nature, and utter weakness of intellect which are both independent of one another. This melancholia may come on suddenly or slowly; it may be, for some time, the only symptom; but it may also be preceded or followed by the intellectual weakness. At the same time there are symptoms of cerebral disease.

The melancholia is generally of a lypemaniac character. In some cases lypemania is accompanied by much anxiety; that is, the patients think only of suicide, ruin, and so forth; in others the lypemania is *stupid*: the patients are foolish and stupid.

The symptoms of cerebral disease that are not of a psychical character are most generally the following. The face has generally a stupid look, brought on by relaxation of the facial muscles, together with redness brought on by a blood-stasis, on which M. Mairet seems to lay much stress. There is also some deviation of the muscles of one side of the face, some defect of symmetry in the mouth, and a little hesitation in the speech. M. Mairet insists very much upon another symptom; that is, on ptosis of the eyelids, either on one or both sides. Other symptoms are to be found in the limbs: formication, feeling of heaviness, for instance; others concern the head: such are vertigo, apoplectiform attacks, cephalagia, heat of the head, and other such. As to the cerebral lesion which M. Mairet considers as the cause of the symptoms alluded to, it is a *localized periencephalitis* of which the seat is the *inferior aspect of the brain*, either on the *whole surface* or on a *limited region of that surface*. M. Mairet considers this disease as brought on, either by propagation of an inflammatory disease of the eye, or ear, or other organ or tissue in the neighbourhood, or by general depressing causes, physical or mental.

Such is the author's general theory, which he supports by many cases with full particulars. But M. Mairet has wished to know whether experiments on animals could in some way support the idea. He has accordingly studied on dogs the effect of certain experimental lesions, observing whether there was some peculiar sadness to be detected on their countenance after the operation. It must be confessed that this part of the book of M. Mairet is much less reliable and valuable than the preceding one. Even if the operation be conducted in the very best manner, it seems very difficult to tell with some degree of certainty whether the animal is "triste" or not. M. Mairet pours upsome water with hydrochloric acid on the inferior aspect of the brain, and watches whether the dog becomes triste. If we judge of the mental condition of the dogs by the sketches M. Mairet gives in his book, they certainly seem very triste; but might they not seem as much so if one had only made a hole in their head without sending any hydrochloric acid into it; or might they not seem exactly the same if the hole

had been made on the superior aspect of the head, and hydrochloric acid poured in, *secundum artem?*

M. Mairet has conducted three experiments on dogs, and concludes that the sphenoidal lobe of the cerebrum seems to be the centre for sadness. We cannot agree with the author's views; three experiments are not enough, and M. Mairet ought to study the influence of other brain-lesions on the *genesis* of sadness, in man as well as in dogs. His book is nevertheless a very interesting and able effort to solve a very difficult problem of brain-function. It is well written, and students of neurology should read it.

HENRY DE VARIGNY.

De la Chute et de la Dystrophie des Ongles chez les Ataxiques. Par E. DOMECQ-TURON. Thèse pour le Doctorat en médecine. Bellier & Cie. Bordeaux, 1883.

THIS is an interesting contribution from the clinical side towards a further knowledge of the trophic lesions of tabes dorsalis. The author has collected fourteen cases—some of them already published by MM. Joffroy, Pitres, Pouget, and Roques, others communicated to him by his friends—which render it highly probable that occasionally loss and deformity of the toe nails are real trophic lesions which should be placed in the same category as the joint affections, and the perforating ulcers of the feet which are not uncommon in the course of this disease. His cases seem to establish the following facts: that loss of the nails is usually limited to the great toes, that it has no fixed relation to any particular period of the disease, and may occur over and over again. The conditions which precede the nail shedding are very variable: without any history of injury the matrix of the nail may sometimes become ecchymosed, and the neighbourhood of the nail may be an æsthetic, or be afflicted with abnormal sensations, or even intense pain. The nail may be reproduced either of normal shape, or more or less deformed. The description, in fact, which the author gives of the nail appearances before shedding reminds one forcibly of the trophic changes which are so often seen when there has been injury to a peripheral nerve, and neuritis is kept up by irritation at the cicatrix. It is this similarity which more especially leads to the supposition that loss of the nails is probably due to a peripheral neuritis rather than to any lesion in the spinal cord, but in any case, it seems clear that spontaneous shedding of toe nails is more often observed in tabes dorsalis than in any other of the degenerative diseases of the marrow.

Not less interesting than loss of the nails are the nail deformities —the *Dystrophie*, as he calls it—of which the author treats in the second half of his thesis. Here again we are reminded how often the nails may become crumpled or misshapen after lesions of peripheral nerves, and it is well known how the nutrition of the nails is dependent on bodily health, and that constitutional illnesses may bear traces of their severity in transverse furrows of all the nails

The dystrophy of the nails in tabes dorsalis, however, is usually limited to the nails of the toes—more especially of the big and little toes—limited, in fact, to the limbs which are most frequently affected with lightning pains or other abnormal sensations. In all the cases recorded the finger-nails presented nothing abnormal. This important association is, moreover, established that at whatever period of the disease the deformity arises, whether it affects the great toe only or the other toes as well, it coincides with the occurrence of severe pains, or with sensations of numbness and creepings in the toes. With regard to the deformity itself, the nails become thickened, elevated or crumpled, they develop longitudinal or transverse furrows, are raised from their natural beds, become intensely hard, and whatever be their colour, lose their natural transparency. In some instances these changes have come on with great rapidity, and once established they seem to last an indefinite time; and as of the spontaneous shedding, so of these dystrophic alterations, the conclusion seems to be that the underlying cause is a peripheral neuritis. The author's thesis is of especial value, because of its well-recorded cases, and the appearances described by him are shown in two plates of coloured drawings. Many observers must be familiar with the kinds of nails depicted, but important results will flow from M. Domecq-Turon's contribution to the clinical history of tabes dorsalis, if the existence of these nail deformities leads to the more careful examination of all patients who present them, and tabes dorsalis be thereby found when least expected, or when, as so often happens, the more prominent symptom of ataxia is entirely absent.

HERBERT W. PAGE.

Critical Digests and Abstracts from Journals.

Report on Visceral Neurology. The Vagus.—The whole subject of the pathological relations of the vagus has been taken up by Kredel, assistant to Professor Riegel in the Medical Clinic of Giessen, and elaborately treated in his inaugural dissertation (*Deut. Arch. f. klin. Med.* xxx., p. 547). The first part of the work touches very lightly on the various diseases and disorders connected with the branches of the vagus to the heart, the larynx, and the lungs proper. The nervous theory of asthma is well summarised; but to this part of the subject we shall immediately return when we consider the researches of Kredel's master, Professor Riegel. Angina pectoris is next dealt with, much after the arrangement of Eulenberg and Guttmann. All these summaries are but an introduction to the special subject of the work, which is devoted to the discussion of two cases under the writer's observation, apparently of an anginal and asthmatic nature combined. As is well known, the respiratory disturbance in true angina pectoris is insignificant, and has been referred, such as it is, to the pain. Kredel contends, however, that a careful perusal of recorded cases reveals the incorrectness of this view; and his own cases, in his opinion, support the belief that the respiratory and cardiac branches of the vagus may be at one and the same time the seat of a severe neurosis. The best-marked case may thus be described. A girl of 16, suffering from mitral incompetence, on being sent to bed for the purpose of examination, was suddenly seized with palpitation and urgent dyspnœa, the pulse rising to 148 and the respirations to 52. Under the writer's observation the dimensions of the pulmonary resonance rapidly increased, along with the size of the heart as tested by the impulse. In the course of half an hour, the urgent symptoms continuing, cough set in with expectoration of blood-stained phlegm, and therewith râles were discovered for the first time in the chest. The dyspnœa, pulmonary dilatation, and cardiac frequency had not entirely disappeared at the end of 18 hours. There was no relapse. Kredel's

interpretation of this attack is that it was a combination of inhibition of the cardiac branch of the vagus, spasm of the bronchi from irritation of the bronchial branches of the vagus, and consequent congestion of the mucous membrane of the respiratory passages. He confesses that the frequency instead of depth of the respiratory acts was not suggestive of true spasmodic asthma, but the acceleration of the pulse and the bronchial catarrh might account for this peculiarity. The whole condition, in a word, was, he believes, a neurosis of the vagus originating in its trunk. He has difficulty in settling—indeed, he confesses he cannot determine—how a lesion of the trunk of the vagus is likely to cause at once paralysis of the cardiac branches and irritation of the pulmonary branches. Unfortunately for Kredel's elaborate theory, we fear that in this country the condition which he observed and has described with such praiseworthy accuracy would be considered to be nothing more than temporary pulmonary congestion of purely mechanical origin.

The neurosal theory of *bronchial asthma*—that is, the relation of disorder of the vagus to this affection—is the subject of a very elaborate investigation, involving a large number of experiments on animals, by Professor Riegel of Giessen, the author of the article on Asthma in Ziemssen's *Handbuch*. The paper (which appears in the *Zeitsch. f. klin. Med.* v., p. 413) commences with an exceedingly valuable *résumé* of the arguments for and against the spasmodic theory proper of asthma. Riegel insists, in the first place, upon our keeping perfectly distinct the two questions as to whether, first, the bronchi contract when the vagus is stimulated, and whether, secondly, bronchial asthma is due to irritation of the vagus. The first question may now be considered as definitely settled in the affirmative—that faradisation of the vagus does increase the pressure within the bronchi, and that this rise of pressure is due to contraction of the bronchial muscles. Riegel's experiments, beginning with this part of the question, were arranged to discover, next, whether the lung increased in size under these circumstances. The result was positive, the dilatation being rapid and considerable, lasting 10 minutes, and slowly disappearing. These observations were made on the undivided vagus, and now came the difficulty—by what route was the impression conveyed which ended in pulmonary dilatation? It was first determined that the irritation did not act peripherally; and the important point appeared to be definitely settled, that, after all, the dilatation of the lungs in asthma is not due to spasmodic contraction of the bronchi. A further series of experiments revealed the fact that irritation of the

vagus produces asthma reflexly, that is, by a centripetal impression which passes through the respiratory centre and reaches the diaphragm, and that the dilatation of the lung is almost entirely due to sudden inspiratory depression of this muscle, followed by its tonic contraction. The theory of bronchial spasm as the cause of asthma must, therefore, be given up as being insufficient. There is unquestionably a rise of pressure in the respiratory passages when the vagus is irritated, as we have seen, but it is much too small to account for the dyspnœa of asthma, and is quite insignificant in comparison with the reflex effect through the diaphragm. Riegel, however, wishes it to be distinctly understood that he does not publish these results as a full and complete explanation of the phenomena of bronchial asthma, inasmuch as vaso-motor disturbances and bronchial hyperæmia are probably elements in the attack.

Respiration.—Some account has been given from time to time in these *Reports* of the discussion (of a thoroughly practical kind) which has been going on in Germany, respecting the physiology of the respiratory branches of the vagus, including the effect of artificial stimulation of the trunk of the nerve in the neck, and of mechanical irritation of its pulmonary roots by respiratory movements. Knoll, who is well known for his important contributions to the physiology of the circulation, has recently published several observations on the effect of electrical and mechanical stimulation of the cervical vagus on the respiration. In the first place, he has shown that modifications of the proper nerve current (*Eigenstrom*) in connection with the transverse section of the central stump of the trunk, can be induced by manipulation, and that these modifications exert a distinct effect upon the respiratory acts in expiratory time. The peripheral stump gives no corresponding reaction. Secondly, Knoll's experiments appear to prove that the primary effect of faradisation of the central stump is expiratory, the more familiar inspiratory effect following stronger stimulation only. But even whilst inducing continuous contraction of the diaphragm in this way, faradisation of the cervical vagus causes a certain amount of simultaneous inhibition of the respiratory movements; for the muscles of extraordinary respiration are relaxed, there is no respiratory movement in response to the cerebral anæmia, and reflex respiratory movements do not follow stimulation of other afferent nerves (*Wien. akad. Sitz.* LXXXV. and LXXXVI.; and *Cent. f. d. med. W.*, 1883, No. 22).

The question of the relative influence upon the respiratory

centre of an excess of carbonic acid and a deficiency of oxygen in the blood, has been revived by Bernstein (Du B. Reymond's *Archiv*, 1882, p. 313: and *Cent. f. d. med. W.* 1883, p. 354). This observer concludes that "carbonic acid dyspnœa" is chiefly expiratory in character: the dyspnœa caused by want of oxygen chiefly inspiratory. It is not until the controlling function of the vagus has been removed, by section of the nerve in the neck, that these points can be demonstrated.

Nervous Influence of Respiration upon the Circulation.—One of the most complex problems in the physiology of the circulation is the effect of respiration upon the movement of the blood in the lungs, the heart, and the systemic vessels. Whilst the phenomena themselves are but imperfectly known, the manner in which they are produced is necessarily still more obscure. Manifestly a great part of the influence of the movements of the chest and lungs upon the circulation is purely mechanical, but there is as unquestionably an abundant nervous connection also between the two great functions. A contribution to this part of the subject has just been made by Taljanzeff (*Cent. f. d. med. W.*, 1883, p. 401), who has discovered that faradisation of the central end of the pulmonary branches of the vagus, divided where they are passing to the (left) lung, near the bronchus, causes great slowing of the heart, increased size of the pulse, and marked fall of the aortic pressure. As these effects can be arrested by section of the corresponding vagus in the neck, the impression manifestly travels to the medulla through this route. It would thus appear that the movements of respiration modify the general blood-pressure through the centripetal branches of the vagus in the lungs.

The Heart.—Perhaps no portion of morbid anatomy is so much a blank as the diseases of the nervous structures of the heart. Dr. Uskow has investigated with care the condition of the nerve-fibres and ganglia in a certain number of instances of enlargement and other morbid states of the heart, and has arrived at the following conclusions (Virchow's *Archiv*, Band 91, III., p. 453). In hypertrophy of the heart the cardiac nerves lose their medullary sheath, and undergo a process of nuclear proliferation. In unilateral hypertrophy of the heart these changes may be found to be confined to the corresponding side. In certain acute diseases the cardiac nerves undergo parenchymatous inflammation. The only changes which Uskow could discover in the nerve-cells was thickening of the capsules and nuclear proliferation, the protoplasm never presenting any appreciable alteration.

Vaso-Motor Nerves.—The vaso-dilator function of the sympathetic is the subject of an elaborate investigation by Dastre and Morat (*Arch. de Physiol. Norm. et Path.* 1882. ix. 2 and 3; and *Cent. f. d. med. Wiss.* 1882, p. 731.) The following are the most important conclusions, as far as our present purpose is concerned. 1. Weak induction currents applied to the upper end of the divided vago-sympathetic trunk in the dog, causes active congestion of the mucous membranes and skin of the same side, with elevated temperature and swelling. The dilatation of the vessels is a direct effect, and not the result of exhaustion of the nerves. It is the sympathetic alone that contains the vaso-dilator fibres, just as it also contains vaso-constrictor, secretory and ciliary (dilator) fibres. 2. The vaso-dilator fibres of the trigeminus (superior maxillary) probably come from the sympathetic. 3. The centre or centres for this function (as regards the mouth) are situated in the dorsal part of the cord (cornua); and the route from the centres to the cervical sympathetic passes through the *rami communicantes* from the 1st to the 5th dorsal. 4. Cerebral conditions, such as emotions, readily influence these nerves. 5. The vaso-dilator nerves of the head are also very amenable to reflex influences, of which those that traverse the vagus are the most powerful: such influences, for example, arise in the outlying regions of the vagus (above all, in the respiratory organs), travel through the medulla and upper cord, pass into the cervical sympathetic, and thus reach the face. 6. Occasionally vaso-constriction and not vaso-dilatation is the result of weak stimulation of the cervical sympathetic; but a different set of nerves are then in action.

Trophic Nerves.—A very elaborate contribution to this subject, of a purely experimental kind, by Lewaschew, of St. Petersburg, working under Professor Botkin, appears to confirm the growing belief that the nutritive changes which follow nervous lesions are referable to vascular disturbance (*Cent. f. d. med. W.*, 1883. p. 193). In one most important respect Lewaschew's conclusion is different from previous views: he holds that the dilatation of the vessels and associated phenomena, consequent on nervous lesions, and frequently the precursors of "trophic" changes in the tissues, are due to irritation of the vaso-dilator nerves, and not to paralysis of the vaso-motor nerves. Lewaschew's investigations consisted in irritating the sciatic nerve in dogs, by means of threads steeped in weak acids or salt, and then noting the phenomena that ensued in the limb, as well as making careful post-mortem examinations of

all the parts involved. Contraction of the vessels of the limb was rarely the result of this operation on the sciatic; and when this result was obtained, no trophic changes ensued. Generally the very opposite condition of circulation was produced: the vessels dilated and pulsated, the limb swelled, the temperature rose; and all in the course of a few days. These phenomena would persist for several months, and then disappear rather quickly, but meanwhile more permanent changes were taking place. The soft parts round the nails were swollen and ulcerated, the epidermis of the sole thickened, the bones became enlarged, the electrical condition of the muscles was altered. Ultimately, the limb generally became atrophied, with diminished temperature; the skin and some of the muscles were reduced in volume; the subcutaneous tissues were sclerosed and white; and the blood-vessels presented contractions and dilatations of their lumen. Microscopical examination confirmed the observation of primary vascular dilatation, and secondary sclerosis around the vessels. In some of the experiments, excess of irritation caused muscular paralysis of the limb, complicating but practically confirming the observation. The absence of paralysis and anæsthesia in the best cases suggested the absence of vaso-motor paralysis, and the presence of vaso-dilator irritation. The variety of results obtained confirmed this view; as well as the rapidity of their development, and the degree to which they advanced.

Two very definite cases of "trophic" or "neuro-paralytic" keratitis may be here recorded. In the one, under the care of Professor Senator, ulceration of the left cornea was associated with trigeminal paralysis, although the "spontaneous" movements of the lids were perfect, as well as the reflex from the opposite cornea. Senator's view is in favour of the "vaso-motor-trophic" origin of such a case. The second case, under Kahler, commenced with paralysis of the right facial nerve only, without keratitis; the lesion being a tumour involving the right ganglion geniculi. As the disease spread, sensibility disappeared in the region of the right trigeminus; and thereupon the cornea very quickly ulcerated, although it had been most carefully protected during the whole illness.

Herpes.—Nieden has described a remarkable case of recurrent herpes in the region of the ophthalmic division of the left trigeminus, after lesion of the cervical spine. A man who had sustained an injury in the neighbourhood of the superior cervical ganglion, and who for the next six years had suffered from

severe attacks of cephalalgia and ciliary neuralgia, became subject to eruptions of herpes in connection with the left eye. The cornea, and the skin corresponding to the supra-orbital and supra-trochlear nerves were the seats of the herpes, whilst the nasal area was spared. During the next six years the eruption recurred four times, and, as in the first attack, it was always accompanied by paresis of the vessels of the left half of the face, periodical headache, hyperæsthesia of the skin, and palpitation of the heart. No doubt the left cervical sympathetic was paralysed, with consequent neurosis of the first division of the trigeminus.

Trophic changes in the skin, hair, or nails were entirely absent in a case of injury to the shoulder and brachial plexus in which there was extensive paralysis of the muscles of the arm, forearm, hand, and chest (Erlenmeyer, *Cent. f. d. med. Wiss.* 1883, p. 112). On the other hand, fresh instances continue to be reported of affections of the toenails in locomotor ataxy. In one case recorded by Roques (*Union méd.* 1882, No. 91) the loss of both great toenails was preceded three months by continued subungual ecchymosis, and the corresponding region of the skin was anæsthetic.

The Stomach.—Prof. Leyden, of Berlin, has published a long and valuable paper upon periodical vomiting of nervous origin, more familiarly known as "gastric crises" (*Zeitsch. f. klin. Med.* iv., p. 605). Whilst most common in association with locomotor ataxy, periodical nervous vomiting may occur in other diseases of the cord, and occasionally appears to be independent of all spinal lesions, although complicated by other nervous symptoms. This statement is, of course, made with full knowledge of the important observation of Charcot that gastric crises may occur as the earliest symptom of ataxy, sometimes even years before the difficulty of locomotion. The onset of these periodical attacks is sudden; and the principal symptoms are severe copious vomiting, epigastric pain, a feeling of severe illness, intense headache, hyperæsthesia of the special senses, and sometimes disorder of the mind. The attack may last two or three days; then a little food is taken; the appetite and digestion are rapidly restored; and in a few days the patient is quite well. As important concomitants of the more striking symptoms, must be mentioned retraction of the abdomen, obstinate constipation, scantiness of the urine, frequency and fulness of pulse without pyrexia, and occasionally severe pains in the limbs. Leyden has twice had an opportunity of making a careful *post-mortem* examination in this class of cases, with entirely negative results as far as the stomach was concerned. The treat-

ment consists in general and local rest, including the use of morphia.

Diabetes and Polyuria.—A case in which the tracts between the "diabetic centre" and the viscera were involved, rather than the centre itself, is recorded by de Jonge in the *Archiv f. Psych.* XIII., 3, p. 658, and *Cent. f. d. med. W.*, 1883, p. 335. A man of 37, with phthisis and tubercular peritonitis, died of sudden right hemiplegia. Post mortem, a bean-sized tumour was found in connexion with the medulla, beginning 0·2 mm. below the left olivary body, extending to the first cervical nerve, and involving the substantia gelatinosa, beyond which it slightly extended on all sides.

A less definite instance of the connection between lesion of the central nervous system and polyuria was observed by Hagenbach (*Jahrb. f. Kind.* N. F. XIX., p. 214; and *Cent. f. d. med. W.*, 1883, p. 352). A child of 4½ years, who had passed at times as much as 10 litres of urine, with steady emaciation and great thirst for nine months, died of tubercular meningitis. Besides the usual post-mortem appearances, a caseous mass was found involving the infundibulum, and a spot of softening in the corpus striatum, with dilatation of the lateral and fourth ventricles.

The Bladder.—The movements of the urinary bladder, and especially the influence of nervous impressions in urination, have been studied by Mosso and Pellacani (*Arch. ital. de biologie*, 1882, i. 1; and *Cent. f. di. med. Wiss.* 1882, p. 705). Besides the movements communicated to the bladder by the respiratory acts, there are purely reflex vesical contractions, which are readily distinguished from the communicated movements by their duration, the shortest of them lasting 6 or 7 seconds. These reflex movements are induced by sensory and psychical stimuli; for instance, a loud noise or a powerful emotion; and, indeed, the authors go so far as to say that "every mental act in man is accompanied by a contraction of the bladder." And not only this, but one can voluntarily empty the bladder without employing the respiratory muscles at all. It is probable that the "voluntary" power referred to in this connection is the power of the will to call up emotions or ideas, which in their turn induce visceral movements such as are familiar in connection with the rectum, sexual organs, and circulatory apparatus. Mosso and Pellacani indeed show that the reflex movements of the bladder correspond closely with those of the arteries, whilst they are quite independent of them. (Cf. 'Brain,' April 1881 and July 1882).

The Uterus.—Frommel has arrived at some novel conclusions

with respect to the movements of the uterus (*Zeitsch. f. Geb. u. Gyn.* viii. p. 205; and *Cent. f. d. med. Wiss.* 1883, p. 418). He finds that these movements are not referable to a uterine centre lying without the organ. Its contractions are spontaneous, and may occur rhythmically in all stages of its development. Disturbances of the circulation have a marked influence upon the uterine movements, compression of the aorta completely arresting them, and compression of the inferior cava producing the same effect, though less quickly. Fall of the body temperature slows the contractions without interfering with their energy; elevation of the body temperature accelerates them, but high fever completely arrests them. The results apply to rabbits only.

Reflex Paralysis.—Several instances are on record of paralysis of a limb in connection with pleurisy, mostly after removal of the exudation, or in consequence of washing out of the pleural cavity. Francotte has recently described a case in which paralysis of the right arm occurred during the spontaneous disappearance of a moderate empyema of the same side. The paralysis was but slight and transitory. In the opinion of Francotte it was reflex in its nature, the seat of irritation being the sensory nerves of the pleura (*Cent. f. d. med. Wiss.* 1883, p. 109).

J. Mitchell Bruce, M.D.

Intermittent Spinal Paralysis of Malarial Origin. By V. P. Gibney, M.D. (*American Journal of Neurology and Psychiatry*, Feb. 1, 1882.)—Two cases are given, occurring in children of 6 and 7 years old, living in New York. In the first case, there were four separate attacks of paralysis, affecting the lower limbs each time, and once the upper limbs also. They were preceded by fever; malaria was suspected in the house where the child lived. Electrical reactions of degeneration, and wasting of some muscles were observed; so that the case simulated infantile paralysis. Recovery took place within times varying from a week to six months, being aided by removal from home and the administration of quinine.

In the second case there were three attacks, each preceded by fever, during which pains in the limbs had been a prominent symptom. Two weeks after the last attack the child had convulsions, and finally died of exhaustion. No post-mortem.

A third case is mentioned, of a child born in a highly malarious place, of a mother who herself was suffering from malaria. The child in its first year had symptoms of ague, in its second an

attack of hemiplegia preceded by chills and fever; when examined by the author more than six weeks later, the hemiplegia had partly recovered, but both arms were tremulous.

In all three cases the father of the patient was intemperate and the mother delicate. The spleen was not enlarged. On one occasion desquamation of the skin was observed.

As causes of the paralysis, the author thinks that he can exclude —lead, from the distribution of the paralysis—hysteria, from the fact that the electrical reactions were abnormal—infantile paralysis, from the complete recovery made from most of the attacks.

Assuming malaria to be the cause, he suggests that the pathology of the disease may consist in a congestion and œdema of the cord, analogous to that of the spleen.

Case of Destructive Lesion of the Cuneus, accompanied by Colour-blindness. By N. E. Brill. (*American Journal of Neurology and Psychiatry*, August 1882.)—A man, ætat. 63, had a sudden attack of coma, lasting two days, followed by incomplete right hemiplegia and right hemianæsthesia. In a few weeks there appeared hyperæsthesia of the right thigh, tremor of the paralysed side, tinnitus, diplopia, and notably colour-blindness. He appears to have been unable to recognise blue or green. There was no limitation of the visual field. No ophthalmoscopic examination was made. There was a certain amount of aphasia, most marked with respect to the names of colours.

Rigidity with tremor set in on the right side, also some tremor of the left thumb. Patient sank in a few weeks more.

Post-mortem.—The large arteries at the base of the brain were found to be atheromatous. There was a well-defined patch of softening on the left side, involving the cortex of the left occipital lobe where it borders on the calcarine fissure, the upper edge of the in gual gyrus, and the lower half of the cuneus. The white sub-lstance was involved to a depth varying from 3½ to 1 centimetre.

Case of three Tumours of the Encephalon. By W. R. Birdsall, M.D. (*Journal of Nervous and Mental Disease*, April 1882.)—Female, ætat. 20. Headache, followed by a chill and fever, six months before admission. Headache mostly occipital ever since. Paralytic symptoms of two months' duration, with the exception of atrophy of left half of tongue, which was said to have always existed. Vision blurred from childhood. Diplopia, and tinnitus (left ear) for three weeks.

On examination, complete facial paralysis, right side; paresis of right sixth nerve. In left hand some athetosis, but not much loss of power: in left leg, considerable lose of power, with increase of tendon-reflex. Double optic neuritis.

Later on, rapid failure of vision, vomiting, dysphagia, increased difficulty of articulation: convulsion of left arm just before death.

Post-mortem.—Tumours were found in the following situations:

(1) In the right hemisphere, encapsuled in the pia-mater overlying the upper half of the ascending frontal convolution, the upper two-thirds of the ascending parietal, the whole superior parietal lobule and part of the inferior. These parts formed a sort of cavity in which the tumour lay: they were thinned by pressure, but not destroyed.

(2) At the junction of the pons with the medulla, upon the lower surface of the cerebellum, and attached to the seventh nerves of the left side. These nerves ran over its lower surface, the facial was not involved, the auditory was attached to it, part of that nerve running through, part being spread over the tumour.

(3) In the medulla oblongata, reaching from the level of the lower third of the fourth ventricle to below the calamus scriptorius, situated nearly in the centre of the medulla, but involving the left side more than the right. The cells of the left hypoglossal nucleus were shrunken and diminished in number. Otherwise the nerve-tissue was for the most part normal, being pushed aside rather than destroyed.

The character of the tumours was that of fibro-sarcoma.

The author remarks that the left hemiparesis can be accounted for by the tumour of the right hemisphere; the tinnitus by the tumour of the left auditory nerve, the atrophy of the left half of the tongue, with the dysphagia and difficulty of articulation by the lesion of the medulla; but for the paralysis in the district of the right facial and right sixth nerves he can assign no cause.

The Paths of the Vasomotor, Sudorific, and Sensory Nerves in the Spinal Cord. By Isaac Ott, M.D. (*Journal of Nervous and Mental Disease*, July 1882.)—These experiments have reference to the nerves of the cat's hind-limbs, and were made on the plan of previous experiments (on the rabbit) by Ludwig and Nicolaides.

If a hemisection of the cord be made in the dorsal region, the feet become vascular: if now the cord be stimulated in the cervical

region, both feet become pale; it is therefore inferred that vaso-motor fibres run in both halves of the cord.

After complete section, stimulation of the cervical region produces no effect; hence it is in the cord, and not in the abdominal sympathetic that the vaso-motor fibres run.

Further, since sweating of both feet follows hemisection of the cord, it is inferred that the sudorific fibres follow the same course as the vaso-motor.

With respect to the sensory fibres, rise of arterial pressure was taken as an index of effective stimulation of them. If the whole cord was divided, irritation of the sciatic nerve produced no effect. If either lateral column was left intact, irritation of the sciatic caused a rise in blood-pressure, which took place even if the grey matter were destroyed. If both lateral columns were divided, no rise in blood-pressure could be obtained by irritating the sciatics. It is therefore inferred that the sensory fibres run in the lateral columns. If a hemisection was made, irritation of the corresponding sciatic had nearly the same effect as irritation of the opposite sciatic; therefore the sensory like the vaso-motor fibres decussate in the cord.

The author's conclusions are:

(1) That the sensory fibres decussate in part in the spinal cord.

(2) That the vaso-motor fibres also do.

(3) That the sudorific fibres follow the vaso-motors and decussate.

(4) That the vaso-motors run in the lateral columns.

On the so-called Family or Hereditary form of Locomotor Ataxia. By Wm. A. Hammond, M.D. (*Journal of Nervous and Mental Disease*, July 1882.)—The author recapitulates the six cases originally reported by Friedreich. Three new cases by Friedreich and one by Carre are alluded to. Twelve new cases are then given. None of all of these were in the author's opinion instances of true locomotor ataxy, meaning by that term primary sclerosis of the columns of Burdach. The symptoms presented in the hereditary disease are weakness (true paralysis?) beginning in the lower limbs, advancing upwards and finally causing embarrassment of speech. Though inco-ordination may be present, the gait is not that of true locomotor ataxy. There are no lancinating pains; indeed pain may be absent altogether. There is no sense of constriction round the waist. There is no loss of cutaneous

sensibility and micturition is natural; the pupils are unaffected. [The patellar tendon reflex where mentioned was abolished.]

The author thinks that the primary lesion is to be found in the medulla, possibly also in the cerebellum. If the spinal cord be implicated (as it certainly was in Friedreich's cases), the posterior columns are not the only, nor even the principal seat of the disease.

Case with Tumours in the Fourth Ventricle, unaccompanied by special Nervous Symptoms. By H. D. Schmidt, M.D. (*Journal of Nervous and Mental Disease*, July 1882.)—The patient, a negro, ætat. 30, who had had secondary syphilis, died of acute pneumonia and pericarditis, without exhibiting any symptoms of intra-cranial tumour.

Post-mortem, there were found:

(1) A lobulated tumour measuring 25 mm. × 13 × 10, attached by a ribbon-like pedicle to the posterior surface of the right restiform body, pressed upon from above by the right tonsil of the cerebellum, and filling up a considerable portion of the fourth ventricle.

(2) A smaller, measuring 8 mm. × 3, situated similarly on the left restiform body, but without a pedicle.

The tumours were very vascular; their microscopic structure was that of gliomata.

The medulla was tough, like india-rubber, but its minute structure was natural.

The author remarks on the symmetry of the tumours, and on the absence of symptoms, though he raises the question whether the pneumonia may not have had something to do with pressure on the pneumogastric nucleus.

Two Cases of Diphtheritic Spinal Ataxia. By Drs. Julius Rudisch and George W. Jacoby. (*Archives of Medicine*, December 1882.)—Two cases are given from the author's own observation illustrating a form of diphtheritic paralysis resembling locomotor ataxy.

Case 1.—Male, ætat. 20. Mild attack of diphtheria; two weeks afterwards, transient paralysis of soft palate; next, weakness of legs, then of hands. In lower limbs, there was some anæsthesia of the soles, ataxic gait, unsteadiness on closing eyes. In upper limbs, retardation of sensation, want of precision in fine movements. Patellar tendon reflex absent. Electro-contractility of

muscles normal. No cerebral symptoms. In rather more than a year, all the symptoms had disappeared, except that the patellar-tendon reflex was still absent. In a year more this also had returned.

Case 2.—Male. Diphtheria, followed in two weeks by paralysis of the palate. In three weeks more, severe shooting-pains in legs and arms; then unsteadiness of gait. When seen, his gait was ataxic, he could not stand with his eyes shut, or walk in the dark. Fingers numb, fine movements of hands difficult. Patellar tendon reflex absent. Action of pupils normal. Electro-contractility of muscles normal. In seven months he completely recovered, and the patellar tendon reflex had returned.

A third case is quoted from Rumpf.

From the symptoms, the authors conclude that the posterior columns were affected, though on reviewing the investigations that have been made into the pathological anatomy of diphtheritic paralysis, it appears that actual lesion of this part has not as yet been found post-mortem.

Case of Myxœdema. By Elizabeth M. Cushier, M.D. (*Archives of Medicine*, Dec. 1882.)—Female, ætat. 57. Her general health had fallen off since nervous shocks in 1861. Within a few years after that time, swelling of the face was noticed, inactivity, slight deafness. When first seen by author (1877), the legs were œdematous, the tissues of the face thickened, the eyelids baggy, suggesting Bright's disease, but there was no albuminuria.

She improved under tonics and diuretics.

After two years there was a return of the œdema in the legs, vesicles formed and became inflamed and ulcerated: the walk was slow and clumsy; she was slow in rising from the chair or lifting her head. No anæsthesia.

The face gradually became more characteristic; the aspect expressionless, the cheeks firm, waxy, rosy pink in colour: the skin round mouth and eyes white and transparent; the lips motionless. Temperature subnormal, pulse slow.

Subsequently the cardiac dulness became much increased in area: the abdomen swelled. She died in June 1882, after an attack of facial dermatitis, and inflammation of the left upper arm.

Post-mortem.—The subcutaneous tissue of the neck seemed infiltrated with transparent material: the heart was hypertrophied, the kidneys rather large. In all these organs there was

seen, microscopically, a swollen state of the connective tissue, and an infiltration with small, spherical, highly refractive bodies. The normal histological elements were atrophied. In the thyroid, which was small and undergoing (in the left lobe) cystic degeneration, it was found that the alveoli were filled with hyaline material and the blood-vessels were infiltrated.

The changes in the spinal cord were as follows:

Vessels.—Their walls were thickened and infiltrated with hyaline substance; the vessels themselves were dilated, and there were numerous small hæmorrhages, many of them old.

Nerve-cells.—These had undergone yellow degeneration, or were atrophied, or had lost their processes. These changes were most marked in the lumbar region: they affected both the anterior and posterior cornua.

White substance.—Showed small spaces filled with hyaline material; here and there the nerve-fibres were atrophied.

The author remarks on the limitation of the mucoid œdema to the skin of the face and neck; and upon the paresis of the lower extremities. This latter she explains by the lesions in the lumbar cord.

The identity of the pathological process in the cord and in the other tissues seems to show that they are not related as cause and effect, but form parts of some general disturbance of nutrition.

Tumour of Pons Varolii. By F. T. Miles, M.D. (*Archives of Medicine*, Oct. 1881.)—Female, ætat. 17. Well-marked cross paralysis. Complete paralysis of left side of face (involving the orbicularis and frontalis), and of the right arm; incomplete paralysis of right leg. Sensation abolished in left half of face, conjunctiva, and nostril; much impaired in the right upper arm, diminished in the right leg. Hearing of left ear impaired. Paralysis of all the muscles of the left eye; inflamation of left cornea.

Post-mortem.—Externally the pons appeared enlarged and nodulated, the left side especially; the peduncles, especially the left, showing the same appearance. On section it was seen there was a growth occupying the left half of the pons, pushing the raphe to the right. J. A. Ormerod.

Ball on Erotomania (*L'Encéphale*, 1883, No. 2).—Professor Ball narrates in detail the history of a typical case of erotomania *proper*, as distinguished from the mental disturbances peripheral in

origin—nymphomania and satyriasis. He recognises the usually accepted views of its essentially centric origin; the chaste and honourable nature of the passion aroused, and frequent association with the nobler qualities of the mind; its affinity to the forms of "Religious Melancholia"; and the tendency of the erotomaniac to pass through a later stage of melancholia, with delusions of persecution, into hopeless dementia.

Luys on the Cerebral Cortex (*L'Encéphale*, 1883, No. 2).—Professor Luys gives the results of his researches into the structure of the cerebral cortex carried on by means of micro-photographic processes similar to what he formerly described in the 'Archives of Physiology' for 1875. He regards his method as likely to reconcile or explain away the discrepant views held by different observers by the substitution of an exact scientific method of delineation in place of the more fallacious individual judgment. On most points of structural detail Prof. Luys is in harmony with the majority of authorities upon the constitution of the cortex; but he differs in one important consideration from all previous writers, for he wholly ignores the usually accepted division of the cortex into five or six laminated zones of nerve-cells, affirming that we might as readily admit fifty or sixty layers into its composition! Whether or not the process advocated by Prof. Luys is likely to explode the usually accepted ideas of cortical lamination based upon the graphic descriptions of Meynert, Lockhart, Clarke and others; or whether the micro-photographic process in its present stage of development can be relied upon for the elimination of the "personal error" due to subjective bias, may be best decided by an examination of the micro-photograph accompanying Prof. Luys' memoir.

Pozzi on Sclerosis of the Cerebral Convolutions (*L'Encéphale*, 1883, No. 2).—A peculiar and hitherto undescribed form of disseminated sclerosis of the cerebral convolutions resulting in atrophy is the theme of this article. The clinical history of the case is unfortunately somewhat defective; but the coarse appearance of the lesions, topography, and histological examination are detailed *in extenso* with illustrations. The worm-eaten aspect of the cortex-to which the author draws attention, was associated with sclerosed prominent patches identical with the form of hypertrophic cirrhosis described by Bourneville and Brückner (Encéphalite tuberculeuse). In the hypertrophied patches the neuroglia gave evidence, by its density, reactions to pigments and histological features, of being the

site of a chronic inflammatory action probably originating in this tissue: the nerve-cells were very sparsely scattered, the pyramidal cells very few in number, and the large "motor cells" almost entirely absent. The neuroglia has likewise infiltrated with a colloid material. In the atrophied and granular patches the condition was similar, with the addition of a still further condensation of the neuroglia and great paucity of the cellular elements. His arguments strongly favour the assumption that the atrophic sclerosis is but a later stage to which the hypertrophied nodules eventually succumb. W. Bevan Lewis.

Bandelette de l'Uncus de l'Hippocampe (*Archives Italiennes de Biologie.* Tome 11, Fasc. 11).—Professor Giacomini draws attention to certain peculiarities observed by him in the brain of man and the higher Quadrumana, a description of which he has failed to discover in the writings of Krause, Henle, Schwalbe, Retzius, and other anatomists, and which concerns a certain appendage to the uncus hippocampi. Upon careful exposure of the hooked termination of the gyrus hippocampi, he invariably finds (in the fresh brain), the uncus traversed by a grey gelatinous band, measuring 1 to 1·2 mm. across. Emerging from the depths of the hippocampal fissure, this band runs more or less obliquely across the inner and upper aspect of the uncus, measuring from its apparent origin, to its termination, from 2 to 3 centimeters. This structure, which is found constant in man, when traced into the fissure of the hippocampus is found to bend suddenly backwards, to be immediately continuous with the fascia dentata. By means of a minute enquiry into the structure of the uncus, and its superficial band, Giacomini shows that both are identical in constitution with that of the cornu Ammonis and its fascia dentata, but owing to the sudden change in direction taken by the superficial band of the uncus, transverse sections of this part afford appearances which are at first sight most deceptive, and have not been fully appreciated by former observers. If, however, sections are taken parallel to the axis of the uncus, this superficial band is found to be constituted of a superficial and deep layer of granules continuous with each other at one extremity, but separated at their other end, and receiving betwixt them at this latter site a series of pyramidal cells, which are but an extension from the pyramidal layer characteristic of the cornu Ammonis. We have therefore in these sections through the uncus a repetition of the layers met with in ordinary transverse sections through Ammon's horn, but beyond

and outside the four layers proper to the horn we have as constituents of the superficial band the following:—

5. A layer of pia mater and blood-vessels separating uncus from its band.

6. Internal molecular layer.

7. Internal granule layer.

8. Intragranular layer, containing pyramidal cells.

9. External granule layer.

10. External molecular layer.

The oblique direction taken by the superficial band of the uncus sufficiently explains many of the anomalous appearances presented by transverse sections some distance in front of the tip of the uncus. The fascia dentata is larger and more voluminous in apes, its free border smooth, and the peculiarities presented by its anterior end are described as they occur in macacus and cercopithecus. In cercopithecus the dentate fascia is disposed as in man, but numerous and closely-packed *globose* cells, like those of the peripheral ganglia take the place of the pyramidal elements in the intragranular stratum. In lower mammals the dentate fascia is more voluminous, especially at its posterior end; and in the dog and sheep its free border is smooth, and resembles closely a convolution. In these animals it terminates at the most anterior part of the gynus hippocampi with a very gentle—scarcely perceptible—bend, over a poorly developed uncus; in fact, the superficial band may here be said to be absent. Tracing the developmental history of the region in man, and the higher mammalia, Giacomini concludes that the ending of the superficial band in the uncus is a characteristic feature of the brain of man and the higher quadrumana, corresponding to the great development in them of the pes hippocampi and its anterior bend or uncus.

Histologie de la Région psycho-motrice chez le Nouveau né. Par MAGALHÃES LEMOS. (*From an Inaugural Dissertation.*)—The author describes the cortex of the paracentral lobule in the new-born infant as being 3 mm. in depth and as divisible macroscopically into seven layers alternately more or less translucent and microscopically constituted as follows:—

1st layer. An interstitial basis substance, containing cells small in size, but possessing relatively large nuclei.

2nd layer is a well-defined stratum of nucleated cells, larger than those of the preceding.

3rd layer. The cells of this layer are more scattered and less in

size than those of the second layer, whilst spherical nuclei, strongly coloured by carmine, are also found here.

4th layer. This layer is formed by a closer approximation of similar cells; the external limit of this layer is not so clearly defined.

6th layer. This layer is characterised by cells of pyramidal form, and of larger dimensions, amongst which are elements similar in structure and distribution to the the preceding layer.

7th layer. Presents no feature peculiar to itself.

It is to the existence and peculiar constitution of the large elements of the sixth layer that the author calls attention. These cells measure ·030μ to ·040μ long, by ·09μ to ·015μ in breath; some scarcely attain this size, but others reach the dimensions of ·65μ by ·020μ. Amongst these are scattered round cells having a diameter of ·020μ to 030μ.[1] The pyramidal cells stain strongly with carmine, and enclose a nucleus ovoid or elliptic in form which in the larger cells may measure ·009μ to 015μ. in long diameter. Of the branches given off by these cells, that from the apex is the most developed; it describes a centrifugal course, and may be followed to a distance of two to five times the length of the cell. Besides the other branches arising from the base of the pyramid, there is a prolongation from the centre of the cell which takes a centripetal direction (towards the medulla). The sixth layer is especially the site of these large pyramidal cells, although they may exceptionally be traced as smaller elements into the fifth or even the fourth layer. They are not disposed in regular series, as in the cornu Ammonis, but as an irregular interrupted formation, forming more or less circumscribed cell aggregations. An examination of the cortex of the internal aspect of the first frontal gyrus reveals the fact that although it is in direct continuation of the paracentral lobule the histological constitution marks it off as a region entirely distinct from the latter. The author briefly discusses the histological structure of the cornu Ammonis in the brain of the new-born infant, and declines to subscribe to the opinion of Prof. Betz, who has stated that "Ammon's horn (in the new-born infant) is already formed, possessing all its characteristic features, and that the cells are as distinctly seen as in the adult brain."

W. W. IRELAND, M.D.

[1] The dimensions are given as in the original memoir; yet the signification of μ being usually read as *micromillimetre* (·001 mm.), there is evidently an error here, and the measurements must be taken as *fractions of a millimetre*. This explanation would accord with already ascertained measurements of the large ganglionic cells of this region in the adult.